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THE MODE OF INFECTION AND ETIOLOGY OF EPIDEMIC POLIOMYELITIS*

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NEW YORK

I

The two problems of uppermost interest in respect to epidemic poliomyelitis are: first, the mode of infection, and second, the nature of the specific microorganism causing the disease. I propose to deal briefly with these two aspects of the subject.

Two views are entertained regarding the mode of infection; according to one the infectious agent of the disease is communicated by personal contact; according to the other, it is conveyed by the stable fly. The differences involved in these two conceptions are fundamental, and hence the practices looking toward prevention of the malady based on them must be wholly different, if they are to achieve the result desired. For that reason it is imperative that the validity of the two doctrines be carefully scrutinized and appraised.

Let us examine first the evidence available bearing on the insect conveyance of the disease. This notion was suggested, in the first place, by the seasonal prevalence of epidemic poliomyelitis, which is predominantly a disease of midsummer and early autumn. However, it is not strictly speaking thus narrowly limited in incidence, since cases occur in the spring and even in the winter months, although they are few in number.

In the second place, the notion is supported by the rural character of some epidemics, as well as by the relatively wide distances which separate many of the cases. In these instances, however, the peculiarities are of degree rather than kind. Epidemics of poliomyelitis prevail also in towns and cities, and cases may be closely associated as well as widely separated.

The notion of insect carriage received for a time the support of experimental evidence, without which it would have remained merely a suggestive possibility; and the wide currency which this notion has

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* Abstract of address delivered before the Philadelphia Pediatric Society, Feb. 9, 1915.

obtained among the laity depends wholly on an imperfect experimental foundation which has now been largely disproved.

At the Congress of Hygiene and Demography held in Washington in 1912, Dr. M. J. Rosenau of the Harvard Medical School announced, as will be recalled, that he had succeeded in communicating experimental poliomyelitis to several monkeys by permitting stable flies to feed first on monkeys inoculated intracerebrally with the poliomyelitic virus, and then on normal monkeys. Although the studies which he reported were then incomplete and he made his announcement in a judiciously tentative fashion, he expressed the opinion that the flies might carry the infective agent, and also that in conveying it from one human being to another an intervening period of time was necessary, during which the virus underwent some change of development within the insect host. This announcement was followed very quickly by a confirmatory one emanating from Anderson and Frost of the U. S. Public Health laboratory in Washington.

No satisfactory explanation of the successful experiments performed by Rosenau and by Anderson and Frost has thus far been offered, since in no other instance have confirmatory results been obtained. The great importance of the subject led immediately to a repetition of the experiments in several laboratories in this country and in certain laboratories abroad, without yielding a single instance of positive infection. Moreover, Anderson and Frost themselves a little later announced that they had failed subsequently to repeat their earlier successful experiments.

Without pursuing this topic further, we may now turn our attention to the other conception, namely, that infection in poliomyelitis is conveyed through personal contact. In considering that view of the mode of infection it is necessary at the outset to have an understanding of the clinical types of poliomyelitis. So long as the affection was conceived of as a frankly paralytic disease in all instances, it was impossible to trace the connection of cases one with another, but once it was determined—as was done by Wickman—that epidemic poliomyelitis assumes non-paralytic and ambulant forms, the subject of the mode of infection was opened up to restudy and to a wholly new interpretation.

The proof of the existence of abortive and ambulant forms of poliomyelitis is not clinical merely, but depends also on laboratory findings. The several laboratory findings may be stated briefly to be the following: the detection of changes in the cerebrospinal fluid; the demonstration of neutralizing immunity principles in the blood; and the determination of the presence of the virus of poliomyelitis on the upper respiratory mucous membrane. The changes in the cerebrospinal

fluid consist of increased cellular content and the presence of globulin; the two conditions may coexist. The new cells are chiefly lymphocytes. The neutralizing properties of the blood depend on the appearance in the serum of immunity principles absent from the normal blood, which correspond to similar immunity principles arising after a frank attack of paralytic poliomyelitis. The virus of the disease has been detected on the nasal and pharyngeal mucous membranes in such quantity and quality as to make possible the communication of poliomyelitis to monkeys. The unmistakable demonstration, both by clinical observation and laboratory tests, of the existence of abortive and ambulant cases of epidemic poliomyelitis—many of which may and do escape detection—indicates how wide, indiscriminate, and unsuspected the distribution of the virus by personal contact may readily become.

But the facts now in hand carry us beyond the distributing power exercised by the frank and abortive ambulant cases, since it can now be affirmed that epidemic poliomyelitis is one of the diseases in which both healthy and chronic carriers of the microbic agent of infection arise. The few healthy carriers thus far detected consist of persons who have been in very close and intimate contact with persons acutely ill with poliomyelitis; for example, the parents of a paralyzed child. The few instances of chronic carriers now known consist of persons who have recovered from an acute attack, but in whom at the expiration of several months the virus has been detected, by animal inoculation, on the upper respiratory mucous membrane. The two classes last mentioned of potential carriers of the infectious agent add materially to the possibility of wide dissemination of the virus.

It is very important, now that the existence of healthy and chronic carriers of the infectious agent of epidemic poliomyelitis is established, that there should not arise undue concern regarding the dangers of conveying the disease, while the fact should be taken into account in devising measures for the prevention of the conveyance. Possessing as we do at present only the imperfect means of animal inoculation for detecting the virus of poliomyelitis, still it appears that the healthy and chronic carriers are not more numerous in this disease than in many other infections of more common occurrence.

Given, therefore, the possibility of the distribution of the virus being effected by, first, the frankly ill, second, the slightly ill, third, healthy, and fourth, chronic carriers, the striking discrepancy noted between the prevalence of cases in small, sparsely populated rural communities and the more thickly populated towns and cities at once disappears.

To sum up the aspect of the subject concerning the mode of infection, we may now state emphatically that the indications are all in

favor of personal communication of the virus. Hence the measures which we seek to put into effect against the introduction and spread of epidemic poliomyelitis should now be based on the conception of the personal factor as paramount, and not on the notion of insect carriage.

II

I turn now to the question of the nature of the virus or micro-organism causing epidemic poliomyelitis. You will recall that the virus is filterable, that is, it passes through the pores of earthenware filters which exclude under similar conditions the ordinary bacteria. We are now acquainted with a score or so of diseases in man and animals definitely proved or believed to be caused by parasites belonging to this filterable class. It is selfevident that these parasites are very minute; and because of their minuteness it has until recently been doubted whether they had actually been viewed under the microscope.

About two years ago, Dr. Noguchi and I announced that cultures of a very minute organism had been obtained which might be regarded as the possible microbic agent of epidemic poliomyelitis. The cultures were derived from the central nervous organs of human beings and monkeys who had had poliomyelitis. As evidence of the nature of the cultures it could then be stated that experimental poliomyelitis had been produced in monkeys by inoculation of the cultures. We have recently confirmed this result in an interesting manner. The culture employed for inoculation had been isolated about eighteen months before and had gone through a number of generations in artificial mediums. It was cultivated finally in a mixture of ascitic fluid and broth, and the fluid carrying large numbers of the micro-organisms—which are of extremely minute size—was injected, in some cases intraspinally, in others intraperitoneally into rhesus monkeys. The injection produced no immediate effect; indeed, a single injection caused no effect whatever. But when the intraperitoneal and intraspinal injections were repeated three or four times, the animals developed paralysis, and the paralyzed animals showed the peculiar histologic changes of the central nervous system indicative of poliomyelitis. In other words, these experiments showed that a culture of the microorganism mentioned, long removed from the nervous tissues, is capable of causing infection of monkeys, and that by this means the symptoms and lesions of epidemic poliomyelitis are produced.

The fact that several inoculations of the culture were required to cause infection agrees with observations previously made by Lewis and myself, that when a subminimal dose of the usual virus of poliomyelitis is injected into monkeys, no effect is produced; but when the subminimal injections are repeated, paralysis may suddenly supervene. The two sets of observations are, therefore, in accord.

SUMMARY

The data which I have had the pleasure of laying before you have led me to believe, first, that the microbic agent of epidemic poliomyelitis is present in the nasal and buccal secretions and is carried by persons, not insects, and communicated by them in such manner as to gain access to the upper respiratory mucous membranes of other persons, among whom a portion, being susceptible to the injurious action of the virus, acquire the infection and develop the disease.

The clinical variety or form of the disease which they develop may be the frankly paralytic, the meningitic, or the abortive and ambulatory in which no severe symptoms whatever appear. But however the persons may be affected, they become potential agents of dissemination of the virus of poliomyelitis, as do a number of healthy persons who have been in intimate contact with those who are ill, and another group of persons who have recovered from an acute attack of poliomyelitis. These several classes of infected or contaminated persons constitute the active means through which the virus is spread and to the control of which sanitary measures designed to prevent epidemics must be directed.

Finally, the virus or microbic agent of epidemic poliomyelitis appears now to have been cultivated and to consist of minute globular bodies, capable of being distinctly viewed under the high powers of the microscope.

LEUKOCYTE COUNTS DURING DIGESTION IN BOTTLE-FED INFANTS*

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It is the consensus of opinion, or belief of the majority of writers, that one of the physiologic causes of leukocytosis is digestion, or ingestion of food. It is the purpose of this paper to show that such is not constantly the case — at least in bottle-fed infants. Most of the text-books speak of physiologic leukocytosis. Some of the standard works on pediatrics state this leukocytosis to be more pronounced in children; a few speak of its being inconstant; and many ignore it entirely.

Yet it is very important whether this is, or is not, the case, especially in the interpretation of the leukocyte count in pathologic conditions.

REVIEW OF THE LITERATURE

The most striking thing in reviewing the literature on the subject is the fact that physiologic variations in leukocytes are seldom taken into account when drawing conclusions in a comparison of counts. Occasionally an attempt to avoid the disturbing factor of digestive leukocytosis in comparative counts is made,[1] for instance, by taking counts from three to five hours after a meal. The fallacy of this will become clear later on.

The first research in digestive leukocytosis was by Moleschott[2] in 1854. Following his work came that of Hirt[3] in 1856; Malassez[4] in 1876; Halla[5] in 1883; Reinecke[6] in 1883. These are all of interest historically, but as practical researches they do not stand because of poor methods of counting and small number of cases.

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* Received for publication Feb. 18, 1915.
* Read before the Philadelphia Pediatric Society, April 13, 1915.

1. Orr: Leukocyte and Differential Counts in Ward and Open Air Treatment, Am. Jour. Med. Sc., 1912, cxliv, p. 238.
2. Moleschott: Ueber das Verhaltnis der farblosen Blutkörperchen zu den farbigen in den verschiedenen Zuständen des Menschen, Wien. med. Wchnschr., 1854, No. 8, p. 113.
3. Hirt: Ueber das numerische Verhältnis zwischen den weissen und roten Blutkörperchen, Arch. f. Anat. u. Physiol., 1856, p. 174.
4. Malassez: Gaz. mêd. de Paris, 1876, No. 25, p. 297.
5. Halla: Ueber den Hämoglobingehalt des Blutes, etc., Ztschr. f. Heilk., 1883, iv, 198.
6. Reinecke: Ueber den Gehalt des Blutes an Körperchen, Virch. Arch. f. path. Anat., 1889, cxviii, 148.

In 1892, Rieder[7] found that seventeen of twenty-five adults gave digestive leukocytosis, and eleven of twelve children; and that the children showed a higher or stronger leukocytosis than adults. He counted before, and only three hours after the midday meal, however, and counted few cells.

In 1892, Schwinge[8] carried out a small research on two adults and two children, examining the blood at two-hour intervals, and concluded that children showed more digestive leukocytosis than adults.

In 1898, Gregor,[9] in nine researches on healthy children, did not always find leukocytosis; very little in breast-fed children, and then only after many hours. In eight of fifteen babies with chronic injury of bowel function he found a distinct leukocytosis with protein supply. He concluded that it is not clear why one child has and another has not digestive leukocytosis: that in most cases after a hunger period protein food causes leukocytosis; that the maximum count is at the noon hours and after the chief meal.

In 1900, Japha[10] examined fourteen reasonably bowel healthy children of whom eight showed at some time after the meal a higher leukocytosis than before. Of these eight, three could be ruled out because of complications, or great irregularity in the counts, leaving only five of the fourteen. He concluded that one must not call every leukocytosis after a meal a digestive leukocytosis, as variations exist regardless of meals; that his work and that of others before him did not prove a digestive leukocytosis; that digestion and assimilation did not hang together; that as leukocytosis was not present under normal conditions one could not make any differential diagnosis in chronic dyspepsia and enteritis.

The same author[11] in 1901, in a few carefully made researches on his own person, came to the conclusion that adults showed a digestive leukocytosis in a limited sense, in that it occurred after the noon meal, at which time there was a periodical increase of the white blood cells regardless of food. He further stated that the rise was not constant and was hindered by constipation. The increase was mostly in the polymorphonuclear cells.

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7. Rieder: Beitrag zur Kenntnis der Leukocytosis und verwandter Zustände des Blutes, Leipsic, 1892, F. C. W. Vogel.

8. Schwinge: Ueber den Hämoglobingehalt, etc., Arch. f. d. ges. Physiol., 1898, lxxiii, 299.

9. Gregor: Untersuchungen über Verdauungs-Leukocytose bei magendarmkranken Säuglingen, Arch. f. Verdauungskrankh., 1898, iii, 387.

10. Japha: Die Leukocyten beim gesunden und kranken Säugling, Jahrb. f. Kinderh., 1900, p. 242.

11. Japha: Ueber Verdauungs-Leukocytose, Deutsch. Aerzt., Zeitung, 1901, iii, 145.

In 1904 Moro[12] examined seven children. He found that digestive leukocytosis was inconstant, although it did occur on the first cow's milk meal to a breast-fed baby, and he considered this a reaction against foreign protein.

In 1911 Wernstedt[13] examined about twenty-seven children. He found that the curves of leukocytes resembled irregular fever curves regardless of whether the infant was fed breast milk, boiled breast milk, cow's milk, or whether it was the first meal of a new-born baby. His charts show that the average of counts after meals were seldom higher than before. He believed that rest or motion, sleep or wakefulness had considerable influence on the count, and his highest counts were on crying children and the lowest on those in sleep or rest.

There have been many researches to determine the cause of digestive leukocytosis.

Hofmeister[14] found that in the intestine of a cat under the influence of food the lymph cells rose in number and that the lymph vessels filled. This was chiefly in the stomach and upper small intestine. Heidenhain,[15] however, thought that in the thoracic duct the cells were too few and the chyle protein too small in amount to account for the supply in the body.

Pohl,[16] in fifty trials on a dog, always obtained digestive leukocytosis (with three exceptions) when protein food was given. He claimed from his examinations that the intestinal veins were richer in white blood cells than the arteries, and believed this to be the source of the cellular increase after the ingestion of food.

Rieder[7] could not demonstrate this, and thinks the increase is due to a chemotaxis phenomenon, the digestive products in the blood causing an attraction.

Muller[17] found that a prolonged hunger period followed by a protein rich meal gave leukocytosis. Burian and Schur[18] also found that an eighteen-hour hunger period followed by a protein meal caused

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12. Moro: Vergleichende Studien über die Verdauungsleukocytose beim Säugling, Arch. f. Kinderh., 1904, xl, 39.
13. Wernstedt: Bidrag till fragen förekomsten af en digestion Leukocytos, etc., Nord. med. Ark., Stockholm, 1910, 3, f, afd. ii, No. 6, p. 1.
14. Hofmeister: Ueber Resorption und Assimilation der Nährstoffe, Arch. f. exper. path. u. Pharmakol., 1886-7, xxii, 306.
15. Heidenhain: Beiträge zur Histologie und Physiologie der Dünndarmschleimhaut, Arch. f. d. ges. Physiol., 1888, xliii, supplement, p. 1.
16. Pohl: Ueber Resorption und Assimilation der Nährstoffe; Die Vermehrung der farblosen Zellen im Blute nach Nahrungsaufnahme, Arch. f. exper. Path. u. Pharmakol., 1889, xxv, 31.
17. Müller: Klinische Beobachtungen über Verdauungs-Leukocytose, Prag. med. Wchnschr., 1890, xv, 213, 228, 239.
18. Burian und Schur: Verdauungsleukocytose und Verdauung, Wien. klin. Wchnschr., 1897, x, 137.

leukocytosis. They could find no relation to nitrogen excretion, and believed the phenomenon not to be one of resorption, but a reaction against injurious foodstuffs.

The results of von Limbeck[19] were similar to those of Burian and Schur, and he also thought constipation hindered the result.

Reinert[20] from his examinations came to the conclusion that the lowest count of the day was in the morning (six to ten), and the highest about four in the afternoon. He thought food made very little difference.

Goodall, Gulland and Paton[21] in 1903, and Goodall and Paton[22] in 1905 did considerable work on dogs and cats. During the course of digestion they simultaneously examined the blood from the mesenteric veins and arteries, and the bone marrow. Their conclusions were that leukocytosis was present after meals, its maximum being at about four hours; that it was both a polymorphonuclear and a lymphatic increase; that the number and variety of cells were the same in the mesenteric arteries and veins, but much increased in the blood from the bone marrow; and they considered this to be the source of the increased number of cells in the blood during digestion. Removal of the spleen did not hinder the production of leukocytosis.

Finkelstein[23] believed it was the whey content and not the protein which determined the production of leukocytosis during digestion. So also Rosenstern,[24] who believed the rise in cells was a reaction analogous to the fever reaction in ingestion of sugar and salts. He examined only ten children, and made his counts only about three to five times a day.

It is interesting to note that Argand and Billard[25] found in examining two rabbits that starvation caused a leukopenia with mononuclear cells predominating which quickly returned to normal on feeding.

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19. Von Limbeck: Klinisches und Experimentelles über die Entzündliche Leukocytose, Ztschr. f. Heilk., 1889, x, 392.

20. Reinert: Die Zählung der Blutkörperchen und ihre Bedeutung fur die Diagnose und Therapie, Leipsic, 1891, F. C. W. Vogel.

21. Goodall, Gulland and Paton: Digestive Leucocytosis in Normal and in Spleenless Dogs, Jour. Physiol., London, 1903, xxx, 1.

22. Goodall and Paton: Digestion Leucocytosis, Source of the Leucocytes, Jour. Physiol., London, 1905, xxxiii, 20.

23. Finkelstein: Verhandlungen der Gesellschaft für Kinderheilkunde, 1906, xxiii, 160.

24. Rosenstern: Ueber alimentäre Leukocytose, Monatschr. f. Kinderh., Leipsic, 1909, viii, 9.

25. Argand and Billiard: Inversion de la formule leucocytaire sous l'influence de l'inanition, Compt. rend. Soc. de Biol., Paris, 1911, lxx, 146.

Richet,[26] in working on a dog, found that uncooked albuminoid matter produced leukocytosis, and cooked did not, even in enormous doses. He concluded that the reaction was due to certain albuminoid substances escaping the action of the digestive juices and entering the blood.

This review of the literature contains most of the original work done on the subject. It does not contain all, as some of the articles were not accessible. It is enough, however, to give us the ideas of the majority of authors and researchers, which may be summarized as follows:

1. In babies digestive leukocytosis is inconstant.
2. In adults digestive leukocytosis is inconstant.
3. A rise occurs in the early afternoon regardless of food.
4. Animals such as dogs, cats and rabbits do show a leukocytosis after food high in protein.
5. The majority of researchers think protein food to be necessary for the production, and a few think the whey and sugar content to be the cause.

It is significant that much of the work has been done on animals.

OBSERVATIONS ON FIFTY CHILDREN

The present report consists of a study of fifty children, on whom over seven hundred blood counts were made.

The method of study pursued was to take a leukocyte count immediately before feeding, immediately after feeding, fifteen minutes after feeding, one-half hour after, one hour after, one and a half hours after, and so on every half-hour until the next feeding. These groups of blood counts (beginning before one feeding, and ending before another) are taken to constitute a "series" and will be spoken of as such. There are 120 of these "series" in this report.

The leukocytes were counted with the low power; four smears were made on the Jappert-Ewing counting chamber; and the contents of two large squares counted on each, making a total of eight large squares.

All matters of technic were carefully watched, and the same pipette and counting chamber used during a series. The toes were found to be the most convenient part of the anatomy for the removal of blood. As far as possible the same amount of pressure was used in extracting blood, and all causes of local congestion avoided. For example, instead of using one toe during a series, different toes were used.

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26. Richet: La réaction leucocytaire: digestion, etc. Presse. méd., 1913, xxi, 539.

The infants on whom this study was made were all bottle-fed, and, while not exactly normal children, were of the type usually seen in a hospital ready for discharge. They were practically all somewhat underweight for their age. With one or two exceptions they could be classed under three groups: "Convalescent Pneumonia," "Convalescent Gastro-Enteritis" and "Feeding." Eight of these babies were examined during one feeding period only, but all the rest had two or more series made, some running as high as twelve or thirteen.

The results of the analysis of these cases are interesting, in that physiological leukocytosis after feeding was not found in most instances, and the opposite condition of leukopenia prevailed. A single rise in leukocytosis after a meal was not considered a digestive increase unless it persisted, as it may be due to other causes. As pointed out by Wernstedt,[13] it may be the result of crying or great muscular activity. Also in judging of the increase or decrease, the mean of the counts after the meal (that is, during the series) was compared to the count before the meal. As examples of what the extraneous sources of increased leukocyte count may be, see illustrative cases — 6, 7, 8 and 9. These cases show that striking a small artery, crying, cold extremities necessitating much pressure to extract the blood, and getting the blood from a place previously punctured, may be the causes of increased leukocyte counts. When such factors as these could be determined, the increase in leukocyte count was not taken to be a digestive one. The frequency with which such things occurred may be judged by the fact that if these were not carefully ruled out, seventy-five of the 120 series would show an increased count during the digestive period, instead of the forty-four (or 28.3 per cent.) shown below.

Of the fifty children, only six, or 12 per cent., constantly showed an increase in the white blood cells during digestion, although sixteen, or 32 per cent., showed at times an increase, and at others a decrease. Contrasted with this, twenty-eight, or 56 per cent., constantly showed a decrease in leukocytes during digestion.

In considering the 120 series, 28.3 per cent. showed leukocytosis and 68.3 per cent. leukopenia. The remaining 3.3 per cent. were so irregular as to be undeterminable.

A study of Charts 1 and 2 will show the amount of leukopenia and when it occurs. The charts represent a summary of the fourth- and third-hour cases, respectively.

The curves, while irregular, clearly show the drop which in fourth-hour cases is greatest at one hour and reaches over 9 per cent., and in the third-hour cases is greatest between one and a half to two and a half hours, and reaches over 11 per cent.

SUMMARY OF CASES

No.	Diagnosis	Age in Months	Gaining + Losing —	Stools During Day	Food	Average Count of Series	Time of Count	After Food, Leukocytosis + Leukopenia —
1	Slight enteritis ..... Underweight.	8	+	Three to six greenish-yellow, soft; few curds.	Casein milk, 3.7-1.6-3, 4 oz. Casein milk, 3.7-1.6-3, 4 oz.	14,325 17,020	9-12 a. m. 9-12 p. m.	— —
2	Normal except for slight anemia.	14	—	One formed yellow.....	4-4.5-3.5. Whole milk, 8 oz. 4-4.5-3.5. Whole milk, 8 oz.	13,700 13,400	9-12 a. m. 9-12 p. m.	+ +
3	Normal convalescent gastro-enteritis.	8	+	Two soft yellow.....	2.6-3-2.3, 7½ oz. ..... 2.6-3-2.3, 7½ oz.	12,025 11,960	? ?	+ +
4	Normal convalescent slight gastro-enteritis.	10	+	Two soft yellow, with few curds. Two soft yellow, with few curds. Two soft yellow, with few curds.	⅔ milk, 4 oz. ..... ⅔ milk, 4 oz. ..... ⅔ milk, 4 oz.	15,500 12,990 13,725	9-12 a. m. 9-12 a. m. 1- 4 p. m.	— — —
5	Feeding ..... Later developed pertussis.	4	Stationary	Three soft yellow.....	2-6-2, 3 oz. ..... 3-6-2, 4 oz.	15,090 21,450	9-12 p. m. 3- 6 p. m.	+ —
6	Normal convalescent gastro-enteritis..	4	+	Soft yellow ..... Constipated, greenish-yellow.	⅔ milk and 3% sugar, 5½ oz. 3-6-2.5, 5½ oz.	14,160 18,420	9-12 p. m. 9-12 a. m.	+ —
7	Feeding ..... Convalescent from sugar injury.	7	+	One soft yellow..... One soft yellow.....	Casein milk with cream, 5 oz., 3.6-1.7-3.04 Casein milk with cream, 5½ oz., 3.6-1.7-3.04	13,700 15,110	9-12 a. m. 12- 3 p. m.	— +
8	Feeding	½ to 1½	Stationary Stationary Stationary + +	Two greenish-yellow, with fine curds. Two greenish-yellow, with fine curds. Three greenish-yellow with fine curds. Four soft brown..... Four soft brown.....	2.5-4-1.5, 2 oz. ..... Whey, cream, 1.3-5.2-1.2 Whey, cream, 1.7-5-1.2, 2½ oz. 2-7-2, 3½ oz. malt soup 2-7-2, 3½ oz. malt soup	14,990 18,160 14,880 15,050 15,040	11- 1 a. m. 11- 1 a. m. 11- 1 a. m. 11:30 a. m.-2 p. m. 2-4:30 p. m.	— — — — —
9	Feeding	8	Stationary Stationary	One loose greenish--yellow, with fine curds. One loose greenish--yellow, with fine curds.	½ milk and 2% dextro maltose, 4 oz. ½ milk and 2% dextro maltose, 4 oz.	15,290 14,570	9-12 a. m. 12- 3 p. m.	+ +
10	Normal convalescent from gastro-enteritis.	8½	+	Three soft yellow.....	3-6-3, 7½ oz.	18,250	9-12 a. m.	—

11	Normal	20	Stationary	One formed yellow.....	Whole milk, 8 oz.	9,660	?	—
			Stationary	One formed yellow.....	Whole milk, 8 oz.	13,750	?	—
12	Normal convalescent from gastro-intestinal indigestion	8½	+	Four soft yellow.....	4.5-4.4-2.57, 7 oz., whey, cream.	12,470	9- 1 a. m.	—
			+	Four soft yellow.....	3-6.5-2.7, 7 oz., milk mixt.	11,500	1- 5 p. m.	—
13	Normal convalescent pneumonia. Developed pertussis.	24	—	0	Whole milk, 8 oz.	8,570	9-12 p. m.	—
			Stationary	0	Whole milk, 8 oz.	10,620	9- 1 a. m.	? (vacillation)
			Stationary	0	Cream wheat, 1 saucerful	10,660	3 p. m.	—
			Stationary	0	Junket, 1 saucerful.....	9,720	7 p. m.	—
			Stationary	0	Whole milk, 2 oz.	8,800	9 p. m.	—
			Stationary	One soft yellow.....	Whole milk, 8 oz., plus cream wheat.	9,080	9- 1 a. m.	+
			Stationary	One soft yellow.....	Whole milk, 8 oz., plus toast.	8,150	1- 5 p. m.	+
			+	One soft yellow.....	Whole milk, 5 oz., cream wheat.	15,030	1- 5 p. m.	? (vacillation)
			+	One soft yellow.....	Whole milk, 8 oz.	13,920	5- 9 p. m.	—
			+	One soft yellow.....	Whole milk, 8 oz.	15,140	9-12 p. m.	+
			Stationary	0	Oatmeal, 1 saucerful.....	18,530	9- 1 a. m.	+
			Stationary	0	Whole milk, 8 oz.	16,380	9-12 p. m.	—
			+	One loose greenish-yellow	Whole milk, 8 oz.	18,400	1- 5 p. m.	+
14	Normal convalescent pneumonia.	24	Stationary	One hard yellow.....	¾ milk, 8 oz.	12,500	1- 5 p. m.	—
			+	One soft yellow.....	Whole milk, 7 oz.	18,200	9- 1 a. m.	+
			+	One soft yellow.....	Whole milk, 8 oz.	15,500	9-12 p. m.	—
			Stationary	One loose greenish-yellow, with fine curds.	Whole milk, 8 oz., and cream wheat.	16,350	9- 1 a. m.	—
15	Otitis media ..... Gastro-intestinal tract O.K.	30	Stationary	Two soft brown.....	Whole milk, 8 oz.	19,300	5- 9 p. m.	—
			Stationary	Two soft brown.....	Whole milk, 8 oz.	18,980	9-12 p. m.	—
16	Convalescent pneumonia	15	Stationary	Two pasty yellow.....	Whole milk, 8 oz.	18,640	1- 5 p. m.	—
			+	Two soft greenish-yellow	Whole milk, 8 oz.	17,780	1- 5 p. m.	+
			+	Two soft greenish-yellow	Whole milk, 8 oz.	18,650	9- 1 a. m.	+
17	Gastro-intestinal indigestion Sugar in urine vomiting. Later cleaned up.	7	—	Three soft yellow.....	3-5.3-2.6, 3 oz., whey, cream.	15,930	9-12 p m	—
			—	Two soft yellow, with fine curds.	0.58-5-1.9, 8 oz., whey, skimmed milk.	18,500	9- 1 a. m.	—
			—	Two soft yellow, with fine curds.	0.58-5-1.9, 8 oz., whey, skimmed milk.	18,110	1- 5 p. m.	+
			—	Two soft yellow, with fine curds.	0.58-5-1.9, 8 oz., whey, skimmed milk.	17,390	5- 9 p. m.	—
			—	Two soft yellow, with fine curds.	0.58-5-1.9, 8 oz., whey, skimmed milk.	18,730	9-12 p. m.	+
18	Feeding, premature.....	3½	Stationary	Two soft yellow.....	2-7-2, 3½ oz. malt soup	8,850	1- 5 p. m.	—
			Stationary	Two soft yellow.....	2-7-2, 3½ oz. malt soup	12,450	5- 9 p. m.	—
			Stationary	Two soft yellow.....	2-7-2, 3½ oz. malt soup	9,540	9-12 p. m.	—

SUMMARY OF CASES—Continued

No.	Diagnosis	Age in Months	Gaining + Losing —	Stools During Day	Food	Average Count of Series	Time of Count	After Food, Leukocytosis + Leukopenia —
19	Feeding	8	+	Three pasty yellow, with fine curds.	3.5-5-3.5, 7 oz.	10,870	9-12 p. m.	—
			+	Three pasty yellow, with fine curds.	3.5-5-3.5, 7 oz.	9,950	12- 3 p. m.	—
			+	One pasty yellow	3.5-5-3.5, 7 oz.	11,800	1- 5 p. m.	—
20	Bronchitis Gastro-intestinal tract normal.	22	+	One constipated yellow	Oatmeal and whole milk, 5 oz.	20,200	9- 1 a. m.	? (vacillation)
			+	One constipated yellow	Egg and toast and whole milk, 8 oz.	18,250	1- 5 p. m.	—
			+	One constipated yellow	Junket, whole milk, 8 oz.	22,490	5- 9 p. m.	? (vacillation)
21	Gastro-intestinal indigestion	15	+	Four soft greenish-yellow, with fine curds.	2.7-3.1-2.5, 8½ oz., milk and soy bean.	15,400	1- 5 p. m.	—
			+	Three soft yellow	2.7-3.1-2.5, 8½ oz., milk and soy bean.	12,700	9- 1 a. m.	—
			+	Three soft yellow	2.7-3.1-2.5, 8½ oz., milk and soy bean.	13,860	1- 5 p. m.	+
			+	One soft yellow, with flie curds.	3.13-3.9-2.8, 8 oz., milk, soy bean.	14,540	9- 1 a. m.	+
			+	One soft yellow, with flie curds.	3.13-3.9-2.8, 8 oz., milk, soy bean.	14,230	1- 5 p. m.	+
			Stationary	One pasty dark green	2.5-1.5-2, 6 oz., casein milk	16,300	1- 5 p. m.	—
			Stationary	One pasty dark green	2.5-1.5-2, 6 oz., casein milk	17,120	9-12 p. m.	—
			+	One soft brown, with fine curds.	2-7-2, malt soup	23,940	9-12 p. m.	—
22	Feeding, tuberculosis (?)	19	+	One soft yellow	Whole milk, 6 oz.	11,400	?	—
23	Normal convalescent gastro-enteritis.	5	Stationary	Two soft yellow	⅔ milk and 3% dextro maltose, 5 oz.	16,630	9-12 a. m.	—
24	Feeding	8	Stationary	Two soft yellow, with casein curds.	2.5-5-2.5, 6 oz.	10,160	9-12 a. m.	—
			Stationary	Two soft yellow, with casein curds.	2.5-5-2.5, 6 oz.	9,880	12- 3 p. m.	—
25	Feeding	5	Stationary	Two loose brown, with mucus.	Water, 5 oz.	7,960	9-12 a. m.	—
			Stationary	Two loose greenish-yellow	No food	9,950	9-12 a. m.	—
26	Feeding	7½	Stationary	Three pasty yellow, with fine curds.	⅔ milk, 6 oz.	12,310	9-12 a. m.	+
			Stationary	Three pasty yellow, with fine curds.	⅔ milk, 6 oz.	12,720	12- 3 p. m.	—

27	Convalescent enteritis	5	—	Two soft greenish-yellow, with some mucus.	⅔ milk and 2% dextro maltose, 4 oz.	11,600	9-12 a. m.	—
			—	Two soft greenish-yellow, with some mucus.	⅔ milk and 2% dextro maltose, 4½ oz.	11,390	12- 3 p. m.	—
		5½	+	Three soft yellow.....	⅔ milk and 2% sugar, 4½ oz.	17,150	9-12 a. m.	—
28	Feeding	3	Stationary	Three spongy yellow.....	½ milk and 3% dextro maltose, 3½ oz.	10,600	9-12 a. m.	—
29	Feeding	5½	—	Three soft yellow, with fine curds.	⅔ milk and 3% dextro maltose, 5 oz.	5,940	9-12 a. m.	+
			—	Three soft yellow, with fine curds.	⅔ milk and 3% dextro maltose, 5 oz.	5,760	12- 3 p. m.	—
30	Feeding	4½	—	One soft yellow, with fine curds.	2.5-1.4-3, 5½ oz., cosein milk.	91,200	9-12 a. m.	+
			+	Two soft yellow.....	2.5-1.4-3, 6½ oz., casein milk.	13,140	9-12 a. m.	+
31	Feeding ..... Slight intoxication?	8	— !!	Three loose yellow, with fine and casein curds.	¾ milk, 6 oz., boiled....	14,980	1- 5 p. m.	++
			—	Four loose greenish-yellow, with fine curds.	2-5-2, 4 oz.	31,470	9-12 a. m.	— No apparent cause for high count.
32	Slight gastro-enteritis	7½	+	Two loose greenish, with mucus.	½ milk and 3% dextro maltose, 5 oz.	8,830	9-12 a. m.	—
33	Feeding ..... Ulceration mouth.	6	—	Two loose yellow	Buttermilk, 5 oz.	11,800	9-12 a. m.	+
			—	Two soft yellow	Buttermilk, 5 oz.	13,980	9-12 a. m.	—
34	Convalescent gastro-enteritis	14	+	Two loose yellow.....	2.5-3.5-3, casein milk and dextro maltose, 5½ oz.	6,390	9-12 a. m.	—
				Two loose yellow.....	2.5-3.5-3, casein milk and dextro maltose, 5½ oz.	6,920	12- 3 p. m.	—
35	Feeding	15½	+	One constipated yellow..	⅔ milk, 5 oz.	13,400	9- 1 a. m.	—
36	Convalescent gastro-enteritis	7½	+	Two soft yellow.....	½ milk and soy bean, 6 oz.	8,810	9- 1 a. m.	—
				Two soft yellow.....	½ milk and soy bean, 6 oz.	8,070	1- 5 p. m.	+
37	Convalescent gastro-enteritis	8	Stationary	One loose greenish-yellow	2-6-3, 8 oz.	23,800	9- 1 a. m.	+ No apparent reason for high count.
			Stationary	One loose greenish-yellow	2-6-3, 8 oz.	20,580	1- 5 p. m.	+
38	Convalescent gastro-enteritis	7	Stationary	Two soft whitish, with fine curds.	⅔ milk, 6 oz.	11,450	1- 5 p. m.	+
			+	Three soft whitish, with fine curds.	⅔ milk 5½ oz.	9,100	1- 5 p. m.	—

SUMMARY OF CASES—Continued

No.	Diagnosis	Age in Months	Gaining + Losing —	Stools During Day	Food	Average Count of Series	Time of Count	After Food, Leukocytosis + Leukopenia —
39	Convalescent gastro-enteritis Slight bronchitis.	6	—	Two soft greenish-yellow, with fine curds.	2.5-4-2.2, 2 oz.	14,790	9- 1 a. m.	—
			—	Two soft greenish-yellow, with fine curds.	2.5-4-2.2, 2 oz.	16,400	1- 5 p. m.	+
40	Normal	15½	+	Two pasty yellow.....	Whole milk, 8 oz.	10,220	9- 1 a. m.	+
41	Normal	26	?	?	Whole milk, 8 oz.	10,080	1- 5 p. m.	—
42	Feeding	17	+	One loose yellow.....	¾ milk, 5 oz.	10,870	1- 5 p. m.	—
			+	One soft yellow.....	¾ milk, 5 oz.	10,680	1- 5 p. m.	—
43	Feeding	11½	Stationary	Three pasty greenish-yellow.	Buttermilk, 4 oz.	5,870	9- 1 a. m.	—
44	Feeding ..... Convalescent essential edema.	19½	+	Two constipated yellow..	Whole milk, 7½ oz.	8,070	9- 1 a. m.	—
45	Feeding	7½	Stationary	Two loose greenish-yellow, with casein curds.	½ milk and soy bean....	16,890	9- 1 a. m.	—
			Stationary	Three constipated yellow	No food	16,850	1- 5 p. m.	—
46	Convalescent enteritis.....	11	Stationary	Two soft yellow.....	¾ milk and 2% dextro maltose, 8 oz.	9,840	9- 1 a. m.	—
47	Feeding	3	+	Soft yellow	No food	15,260	9-12 a. m.	—
			Stationary	Soft yellow	?, i. e., food unknown...	14,400	9-12 a. m.	—
48	Feeding ..... Convalescent gastro-enteritis.	9½	+	Three soft yellow.....	¾ milk, 7 oz.	14,920	9- 1 a. m.	—
			+	Three soft yellow.....	¾ milk, 7 oz.	13,930	1- 5 p. m.	—
49	Feeding ..... Bronchitis.	5	Stationary	Three loose greenish-yellow.	⅔ milk, 4 oz.	12,640	9-12 a. m.	—
			Stationary	Three loose greenish-yellow.	⅔ milk, 4 oz.	12,760	9-12 a. m.	—
50	Normal convalescent enteritis.	7	Stationary	Two soft yellow, with fine curds.	2.5-4-2, 7 oz.	12,430	9- 1 a. m.	—
			Stationary	Two soft yellow, with fine curds.	2.5-4-2, 7 oz.	10,270	1- 5 p. m.	—
			+	Two soft yellow.....	No food	9,260	9- 1 a. m.	—

The explanation of the fall after the third hour in Chart 1 lies in the fact that this curve, being a composite one of all fourth-hour cases, represents series taken at different times of the day. If we take into consideration the fact that the highest count of the day is before the morning feeding, we have a ready explanation of this peculiar fall.

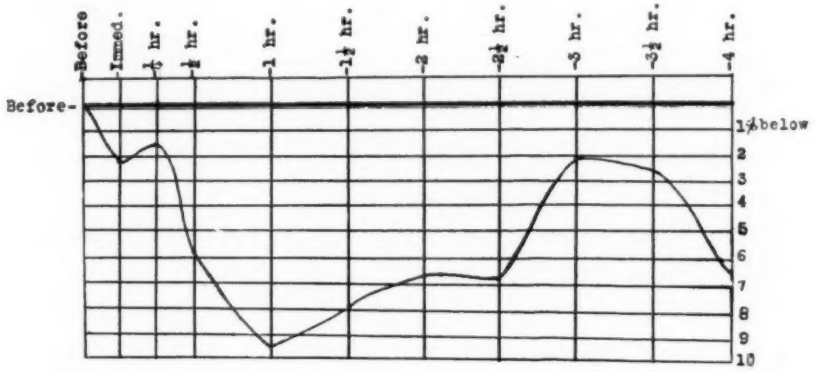

Chart 1.—Fourth-hour cases. In this and the following chart the vertical lines represent time and the horizontal percentages.

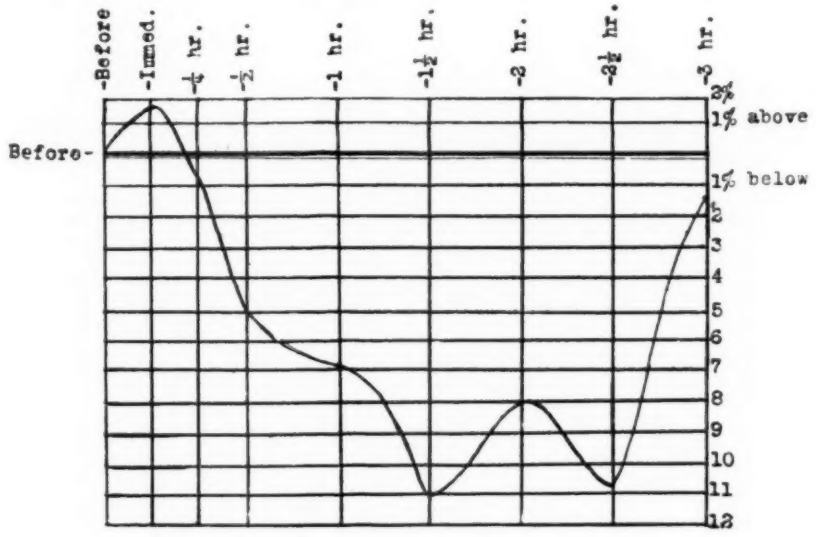

Chart 2.—Third-hour cases.

As this is not marked until about four or five hours after the first morning feeding, it affects the third-hour cases but little. This does not correspond to the findings of Gregor,[9] Japha[11] and others. Of twenty-two cases in the present report which were studied both in morning and afternoon, however, ten showed a higher average of

counts in the morning, eight showed about the same both morning and afternoon, and only four showed a higher average of counts in the afternoon.

Age.—The patients varied from 3 weeks to 26 months in age. No relation of digestive leukocytosis or leukopenia could be established according to age grouping.

Nature of Food.—The majority of the patients were fed on cow's milk mixtures, although a few had casein milk, malt soup, whey and cream, and buttermilk mixtures. The number of patients fed on other than cow's milk mixtures was too small to permit judgment of their relation to leukocytosis or leukopenia.

Temperature.—No relation between leukocytosis and leukopenia, and temperature could be established.

Gain or Loss in Weight.—Of the 120 series at the time of counting, fifty-six, or 46.66 per cent., were gaining in weight; nineteen, or 15.83 per cent., were losing, and forty-five, or 37.5 per cent., were stationary in weight. The proportions of these three groups showing digestive leukocytosis or leukopenia may be shown in the following tabulation:

	Leukocytosis Per Cent.	Leukopenia Per Cent.	No Change or Vacillation Obscured Per Cent.
Gaining (56 series).....	26.78	66.07	7.14
Losing (19 series)	47.36	52.63	0
Stationary (45 series)	22.22	75.55	2.22

Briefly, then, these percentages show that leukocytosis during digestion occurred about twice as frequently in children losing weight as in those gaining or stationary, and that leukopenia is somewhat more constant in children gaining or stationary than in those losing weight.

Time of Day.—The relation to the time of day is shown in the following tabulation:

	Leukocytosis Per Cent.	Leukopenia Per Cent.	No Change Per Cent.
Morning or first feeding (51 series)	27.45	68.62	3.92
Early afternoon or second feeding (39 series)	33.33	64.1	2.56
Late afternoon or third feeding (8 series)	(All showed leukopenia)		
Last feeding or 9 p. m. feeding (18 series)	27.7	66.6	5.5

There does not, therefore, seem to be much relation to the time of day, unless we consider the small number of series taken during the late afternoon feeding.

ILLUSTRATIVE CASES

The counts do not show a regular progressive decrease or increase as the case may be, but are often quite changeable and variable.

For example:

CASE 1.—Showing counts taken during four-hour period, the child receiving no food for several hours before or during the series.

At 9 a. m., white blood cells, 11,000; 9:30 a. m., 10,150; 10 a. m., 9,300; 10:30 a. m., 7,925; 11 a. m., 10,575; 11:30 a. m., 8,540; 12 noon, 7,725; 1 p. m., 8,900. This same child had shown on two other occasions a leukopenia following ingestion of food.

CASE 2.—A child convalescent from acute gastro-enteritis. He is now 13 months old, having normal temperature, and one or two firm, yellow bowel movements a day. Showing leukopenia.

At 9 a. m., white blood cells were 8,475; at 9:02 he was given 5½ ounces of casein milk with 2 per cent. sugar added (2.5-3.5-3); he finished his bottle at 9:15, and blood taken at that time showed a count of 5,875; a quarter of an hour later, white blood cells 6,825; half an hour later, 5,350; one hour, 4,900; one and one-half hours, 6,225; two hours, 5,475; two and one-half hours, 7,125; three hours, 7,275.

CASE 3.—The patient is a child of 1 year, convalescent from an acute gastro-intestinal upset. He is now gaining and having good stools.

At 9:20 a. m., his leukocytes were 6,600; at 9:24, he was given a buttermilk mixture of which he took 4 ounces, and finished this at 9:32. At that time his leukocytes were 5,800; a quarter of an hour later they were 5,700; half an hour, 6,150; one hour, 5,600; one and a half hours, 5,000; two hours, 5,950; two and one-half hours, 5,800; three hours, 6,800; three and three-fourth hours, 5,250.

CASE 4.—A child of 20 months, whose diagnosis was essential edema, but who at the present time is an entirely normal child.

Before his feeding of 7½ ounces of whole milk, his leukocytes were 9,550; half an hour after finishing this bottle, they were 8,600; one hour, 6,600; one and a half hours, 7,360; two hours, 6,300; two and one-half hours, 8,060; three hours, 7,960; three and three-fourth hours, 10,130.

CASE 5.—A child of 8 months, who is not doing very well at the time of examination, as she is having loose, greenish-yellow bowel movements and is vomiting somewhat. She shows a distinct leukocytosis after food.

Before food her count was 18,625. She was fed 8 ounces of a 2-6-3 milk mixture, and immediately regurgitated a small amount. A quarter hour after food her white blood cells were 32,050; half an hour, 28,350; one hour, 25,500; one and a half hours, 24,300; two hours, 23,650; two and one-half hours, 25,800; three hours, 21,850; three and one-half hours, 19,200; four hours, 18,700.

CASE 6.—Showing effect of cold. Before food, 16,675. Fed 6 ounces milk, 3 ounces soy bean gruel. One-half hour afterward, 13,825; one and one-half hours, 17,325 (toes very cold from exposure); two hours, 13,875 (toes warm again); three and one-half hours, 15,275.

CASE 7.—Showing the effect of removing blood from a puncture made some time previously. Before food, 10,425. Fed 6 ounces milk, 2 ounces soy bean gruel. Quarter hour afterward, 13,850; half an hour, 14,675; one hour, 14,400; two hours, 20,950 (blood removed from puncture made at one hour previously), three hours, 12,950.

CASE 8.—Showing the effect of crying. Before food, 10,250. Fed 8 ounces whole milk and two crackers; a quarter of an hour afterward, 9,125; half an hour, 12,000 (crying and struggling); one hour, 10,350; one and a half hours, 7,865; two hours, 9,525; two and one-half hours, 9,415; three hours, 10,600; three and one-half hours, 11,275; four hours, 10,475.

CASE 9.—Showing the effect of arterial bleeding. Before, 11,050. Fed 3-5-3 mixture, 8 ounces; a quarter of an hour afterward, 10,000; half an hour, 9,750; one hour, 10,500; one and a half hours, 12,300 (struck small artery in big toe); two hours, 8,800; two and one-half hours, 8,050; three hours, 8,250; three hours and fifty minutes, 8,700.

CASE 10.—Child showing distinct leukocytosis after ingestion of food. Before, 9,800. Fed ⅔ milk, 6 ounces. Immediately after taking, 9,250; a quarter of an hour afterward, 11,600; half an hour, 10,500; three quarters of an hour, 12,300; one hour, 12,600; one and a half hours, 11,450; two hours, 12,100; two and one-half hours, 11,550; three hours, 11,600; three and one-half hours, 14,200; three and three-fourths hours, 9,900.

CASE 11.—Showing a child followed directly through morning and afternoon feedings, and having a slight but not continued rise after the second meal of the day. Before first morning feeding, 9 a. m., white blood cells, 12,700. Fed 3 ounces milk and 3 ounces soy bean gruel. Quarter hour afterward, 7,625; half an hour, 9,925; one hour, 7,100; one and a half hours, 8,600; two hours, 9,050; two and one-half hours, 7,975; three hours, 8,275; four hours, 8,050. Then fed 3 ounces milk and 3 ounces soy bean gruel. Immediately afterward the white blood cells were 9,025; half an hour afterward, 8,275; one hour, 7,500; two hours, 7,625; two and one-half hours, 7,925; three hours, 7,400; three and one-half hours, 7,350; four hours, 9,500.

A summary of the actual counts on uncomplicated cases shows the following figures for different ages:

Age	Average	Minimum	Maximum	No. Counts
1- 4 months	13,870	8,850	15,290	78
4- 8 months	12,360	7,960	17,150	254
8-12 months	12,290	9,880	15,500	115
12-18 months	12,820	6,390	17,120	103
18-24 months	10,720	8,070	13,750	21
Over 24 months.....	11,520	8,150	16,350	62

Remember these counts are on bottle-fed babies. Carstanjen,[27] in a number of counts, claims the average to be 12,500 in the first year. Rabinowitsch[28] thinks the counts usually given are too high, and says that healthy children from 1 to 16 years should average from 6,000 to 7,000.

CAUSE

As to why some children show leukocytosis and others leukopenia after food, and why the same child may show at times an increase and at times a decrease after food — this research offers no adequate explanation. Careful study of the cases as regards age, gain or loss in weight, character of stools, nature of food, temperature, and time of day did not show any definite relation. It might be added in this connection that six children were studied through a four-hour period,

27. Carstanjen: Wie verhalten sich die procentischen Vehrhältnisse der verschiedenen Formen der weissen Blutkörperchen beim Menschen unter normalen Umständen? Jahrb. f. Kinderh., 1900, lii, 333.

28. Rabinowitsch: Die Leukocyten verschiedenen Altersstufen: Untersuchungen über die Leucocyten gesunder Kinder, Arch. f. Kinderh., 1912, lix, 161.

beginning at 9 a. m., and received no food then or for several hours before. These all showed a gradually decreasing leukocyte count.

A theory which may be offered here is that ingestion of food, and beginning of the activities of the gastro-intestinal tract, both glandular secretion and muscular movement thus causing increased blood supply to the splanchnic area, may attract leukocytes away from the superficial blood, and result in a diminished number of cells when counted by the ordinary methods.

CONCLUSIONS

1. Bottle-fed babies do not constantly show digestive leukocytosis; in fact, the majority show a smaller number of leukocytes in the superficial blood after taking food than before.

2. This decrease is greatest at from one to two and a half hours after food, and tends to rise before the next feeding.

3. When a rise does occur, it is most frequently soon after feeding, and begins to decline in a half-hour.

4. Crying, struggling and chilling of the part from which the blood is extracted increases the count.

5. There seems to be as yet no adequate explanation for the increase or decrease.

6. Comparative counts should be made at the same time of day, and at the same time in relation to food.

I wish to thank Dr. J. C. Gittings and Dr. William Pepper for their valuable advice in the preparation of this work; Dr. Theodore LeBoutillier, Dr. John F. Sinclair and Dr. Arthur Newlin for their kindness in allowing me the use of their cases at the Babies' Hospital.

1805 Pine Street.

THE INFLUENCE OF MENSTRUATION ON BREAST MILK *

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If one reviews the current literature as presented in the text-books on the effect of menstruation on breast milk he is struck with the fact that opinions are very diverse and that many statements are made without sufficient scientific foundation. In an endeavor to obtain scientific facts on this subject we quite carefully looked through the literature and were able to find only a few articles of any scientific value.

In 1853 Vernois and Becquerel[1] in three cases examined the milk secreted before and during menstruation. They found that taken as a whole there was a lower specific gravity with a decrease of water and sugar content and an increasing total solid and casein, while the fats and ash remained about the same during the menstrual period. But a closer inspection of their cases shows that while Cases 1 and 3 agreed with these findings, Case 2 showed opposite results and that whereas the average obtained was as above stated, the individual differences were such as to render the average findings of little value.

Very interesting to American physicians will be the fact that the next article, appearing in 1856, was by N. S. Davis.[2] His examination in one case showed a marked diminution of all the solid constituents of the milk, but here, too, circumstances are such as to make one question the scientific value of these findings, because, while the first examination was made in the intermenstrual period at the end of the seventh month of lactation, the comparative examination made during menstruation was in the thirteenth month of lactation, a comparison which would hardly seem, in the light of our present knowledge, to form a basis for strict scientific deductions.

More recent writers, among them Pfeiffer,[3] Schlichter,[4] and Bamberg,[5] have been unable to note any changes in the chemical composition of the breast milk during the menstrual period which were greater than individual variations which might occur at any time during the

* Received for publication Feb. 3, 1915.

1. Vernois and Becquerel: Ann. d'hyg. pub. et de méd. légale, 1853 (Series 1), lxix, 257 and L, 43.
2. Davis, N. S.: Northwestern Med. and Surg. Jour., 1856, v, 535.
3. Pfeiffer: Jahrb. f. Kinderh., 1883, xx, 359 (392).
4. Schlichter: Wien. klin. Wchnschr., 1889, ii, 978, 1004.
5. Bamberg: Ztschr. f. Kinderh., 1913, vi, 430.

course of lactation. Thiemich,[6] in his review of the subject, upholds these writers.

Our report, however, has nothing to do with the chemical composition of the breast milk, but rather with the influence of menstruation on the quantity of milk secreted. We were not able to find any definite data in the literature on this subject. Schlichter, however, states that the average daily gain in weight of the child during menstruation was 6 gm. higher than before that period.

REPORT OF CASE

As mentioned in a previous paper,[7] the baby with a harelip and cleft palate was born on Feb. 26, 1913, after a pregnancy of eight months. According to the hospital regulations at that time the baby was put to the breast every four hours during the first day and after that every three hours for the first week. During the first week he lost 18 ounces. In the second week the milk obtained by the baby was supplemented with breast milk obtained from other women in the hospital. For a few days before the mother and baby left the hospital an attempt was made to make the baby gain in weight by feeding him every two hours. He did gain 4 ounces.

After leaving the hospital on the fifteenth day the baby was given six feedings of 2 ounces each of modified certified milk, half and half, with one teaspoonful of dextrimaltose. In three days he had lost 4 ounces. We increased the feedings to 3 ounces and at the same time began to experiment with the breast pump. In about two days we put together a pump such as described in the other paper and were able to feed the baby with one-half breast milk and one-half modified certified. Inside of a week the baby was fed with six feedings of 3 ounces each of breast milk and began to gain in weight. When a month old he weighed 6 pounds and 3 ounces. From that time on he gained very regularly in weight.

We fed him regularly at four hour intervals beginning at 6 a. m. during the first two months, increasing the amount of each feeding to 3½ ounces during the fifth week and 4 ounces during the eighth week. After the second month we fed him five times daily, 6 a. m., 10 a. m., 2 p. m., 6 p. m. and 12 p. m., increasing the feedings to 4½ ounces during the tenth week, 5 ounces during the eleventh week, 5½ ounces during the thirteenth week, 6 ounces during the eighteenth week, 6¼ during the twenty-second week, 6½ at the end of the six months and as much as we could pump after the seventh month. The first two feedings in the morning were the easiest to obtain, and of the other

6. Thiemich: Monatschr. f. Geburtsh. u. Gynäk., 1898, viii, 659.
7. Caldwell, Fred C.: An Effective Breast Pump, page 381, this issue.

three feedings, the 6 p. m. feeding was the most difficult to pump and the least in amount.

When the baby was 6 weeks old the mother's menstrual flow returned and after a few months we noticed that in spite of effort we got a smaller amount of milk at the last three of the daily feedings for about one week before the menstrual flow began. Within two days after the menstrual flow began the amount became normal and in a few days was greater than before that period. This occurrence was so regular that we knew that a menstrual period was coming when the daily total amount of milk began to fail.

The only constant effect of the menstrual period on the baby was that he did not gain so much in weight during the ten days preceding the period as he did the ten days following the period. Sometimes he cried a little more than usual or was a little fretful before the period began, and sometimes during the same time he had a few extra bowel movements with a slight greenish color, but these changes were inconstant.

Our records are most complete for the period which occurred in September, 1913, when the baby was about 7 months old. A careful study of this portion of the record will reveal all of the changes which took place. To get a true picture we will begin ten days before the day on which the menstrual flow began and end with the tenth day after the day.

A glance at the weight curve will show that, with the exception of the first three weeks, for the period of nine months the child showed a perfectly normal gain in weight. As shown in the history, the period of the first three weeks was one in which we were endeavoring to find a means of keeping the child on breast milk. After the institution of the breast pump, as shown in the preceding article, the child went along in an altogether normal manner without any nutritional setbacks.

A glance at the curve showing the quantity of breast milk secreted during four successive menstrual periods is interesting for the following reasons: First, in each instance the occurrence of menstruation marked the beginning of an increased amount of breast milk. In the last three periods this increase was steady and reached its highest point on the thirteenth, eleventh and sixth day, respectively, following the beginning of menstruation. Not only is this true, but it is also true that for several days previous to the occurrence of menstruation there was a distinct reduction in the quantity of breast milk. The first period does not show at what day this reached its lowest point, but in the succeeding three periods we find that the smallest amount of breast milk was secreted respectively on the seventh, sixth and fourth day preceding menstruation and that in the last period there had been a drop

which reached its lowest point on the seventh day previous, showing a distinct tendency in this case toward a decrease in the breast milk, which decrease reached its lowest point about a week previous to the occurrence of the menstrual flow.

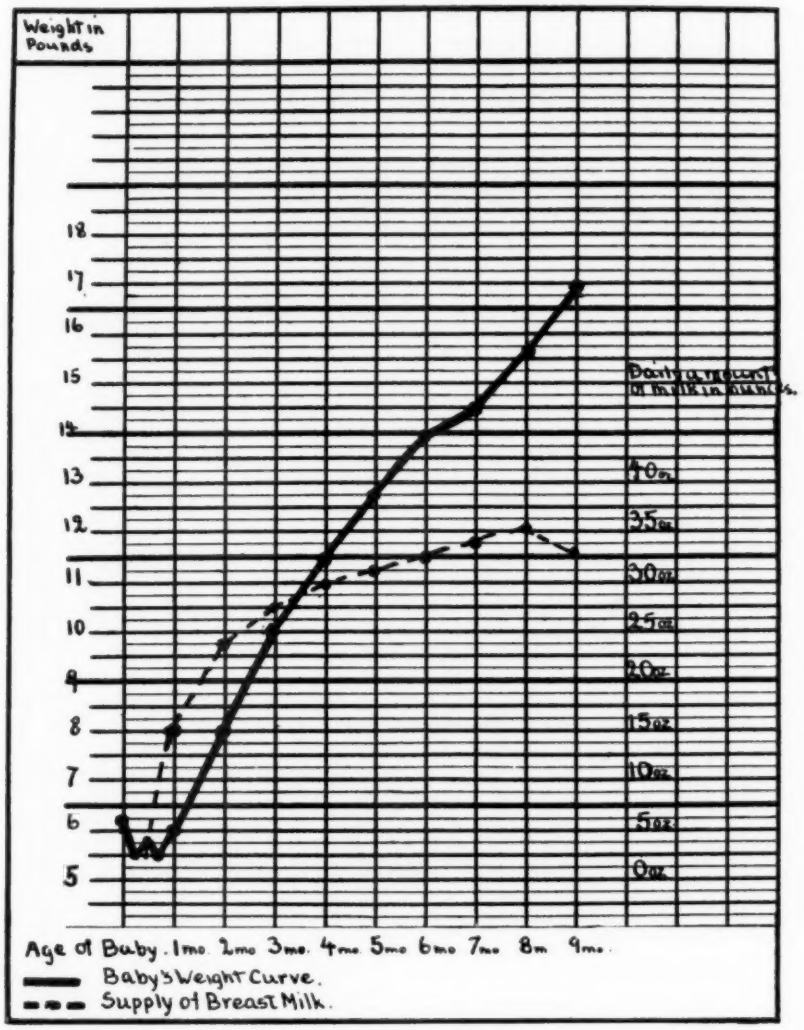

Fig. 1.—Weight curve and amount of breast milk secreted.

The most characteristic months would seem to be the seventh and eighth. In the sixth month the change in the time of the feedings brought about a disturbance in the curve which can readily be seen and which, of course, is not to be reckoned with in this procedure. It is also evident that after the operation, which occurred on November 31,

the regulation in the quantity of breast milk was not the same as it had been previous to that time, and as the first weight curve shows, the total average daily quantity of milk secreted during the ninth month fell below that of the previous two months showing that the breast was probably beginning to give out.

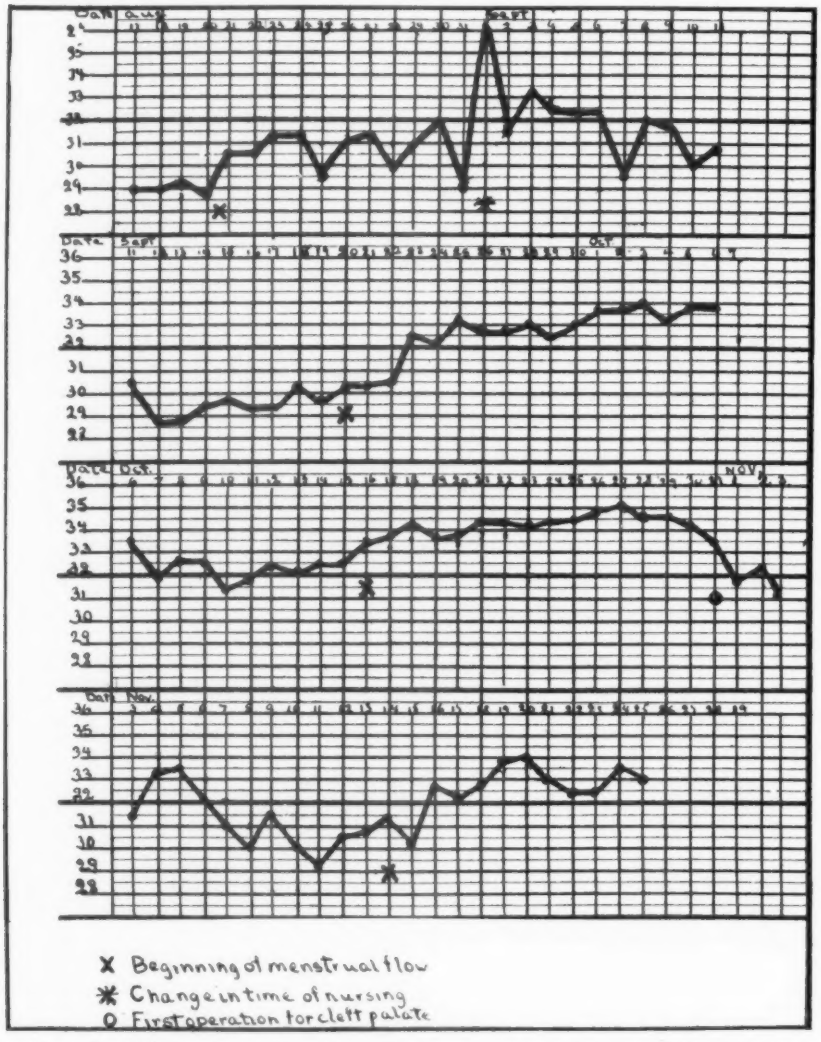

Fig. 2.—Effect of menstruation on the amount of breast milk secreted.

One may say in criticism of the method employed that natural conditions were not present and that therefore these data are of little or no significance. While one cannot deny, of course, that the conditions might have been different had the child been put to the breast, at the

same time there are several things which lead us to think that the method employed does not give us circumstances materially different from those which surround the normal nursing infant. In the first place, while the act of nursing in the young infant certainly shows little or no suction, in the infant which has reached the age of 5 or 6 months there can be no question that the sucking influence is a great, if not the greatest, factor in the withdrawal of the milk from the breast. With the breast pump employed, this suction is exerted in a manner not unlike that used by the infant. In the second place, by using this pump on the wet nurse we have been able to keep up the flow of milk for several months without any apparent tendency to decrease other than might be encountered under normal conditions and have procured in all instances a very satisfactory quantity.

The second objection that may be entered is that the psychic influence would affect the mother in such a way that her efforts to withdraw the milk would be reduced. While the psychic influence is unquestionably of great importance in the flow of breast milk, it seems to us that it would not act under these circumstances in a manner any different from normal. In fact, the period when one would expect the psychic influence to be most marked is that period just previous to and at the beginning of menstruation. In all instances at this time there was a distinct tendency toward an increase in the amount of milk. Since we know so little about the true character of menstruation and the various changes in the physiology of the body which produce it, it seems at this time quite impossible to account for any such variation as is here mentioned.

One other question presents itself and that is that the occurrence of an increased number of stools and a slight tendency to dyspepsia of the breast fed infant during the menstruation period has been too often repeated to be passed by slightingly. The only explanation which we could offer for any such occurrence is that during the premenstrual period the quantity of milk is such as to cover amply the needs of the child; in other words, that the maximum amount (von Pirquet) of food is being consumed, and that during and immediately after the menstrual period the increase in the quantity of milk produces a slight degree of dyspepsia.

SUMMARY

We have to do here with a baby born with a harelip and cleft palate, nursed for nine months by means of a specially devised breast pump. The mother's menstrual period began six weeks after birth and continued throughout the nursing. The quantity of milk was carefully measured, especially during the last four months of lactation, and there was shown a distinct relation between the quantity of breast milk and

the occurrence of the menstrual period. This consisted in a period of increase of breast milk beginning with the first day of menstruation and lasting from ten days to two weeks thereafter. There then occurred a diminution in the quantity which reached its lowest point four to seven days previous to menstruation after which there was a gradual increase.

122 South Michigan Avenue—35 South Dearborn Street.

AN EFFECTIVE BREAST PUMP*

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CHICAGO

On Feb. 26, 1913, an eight-month baby, with a harelip and cleft palate, was born in the Presbyterian Hospital. Because of the defects in the mouth and his general weakness, the baby was unable to nurse from the breast. He was able to get some milk through a nipple shield if the mother massaged the breast at the same time, but he made such feeble suction that in two weeks the supply of milk amounted to only 2 ounces a day. In the first week the baby lost 18½ ounces, and during the second week, when we supplemented the mother's supply by securing milk from other women who had an overbundance, the baby gained 5 ounces. After leaving the hospital on the fifteenth day, we tried to supplement the breast feedings with certified cow's milk and the baby lost more than he had gained during the second week. I concluded that only breast milk could keep the baby alive.

In desperation I had the father use a nipple shield to stimulate the breasts whenever he was at home, and tried all of the breast pumps I could find. When I could not secure a pump that would enable us to get more than 1 or 2 ounces a day, I tried to make one. After a few days of experimenting, I put together the pump shown in the diagram (Fig. 1).

By using this pump the supply of breast milk was increased so that within a week the baby received 18 ounces a day. The pump was used continuously until December 1 without any other means for stimulation of the breasts. During this time the baby gained 12½ pounds. The increase in the amount of breast milk was gradual but steady, and at no time did we have to give a supplementary feeding. At the end of the eighth month the supply was its highest (35 ounces). The baby had nothing but breast milk and, of course, water.

The chart (Fig. 2) and table show the increase in milk supply and the baby's gain in weight.

MATERIALS NECESSARY

1. A glass T tube made of tubing three-eighths inch in diameter.

2. An ordinary glass nipple shield. One of the shape shown has given the best satisfaction in that there is less obstruction to the flow of the milk.

* Received for publication Feb. 3, 1915.

3. Medium heavy glass tubing three-eighths inch in diameter for the suction tips such as shown in the diagram. These can be easily made by any one who has had a little experience in a chemical laboratory. Two of them can be made from a 10-inch piece of tubing. Heat as narrow a ring as possible in the center of the tube until the glass just softens. Pull very gently until there is a slight constriction in the tube. Then after allowing the glass to cool partially, heat the tube again about one

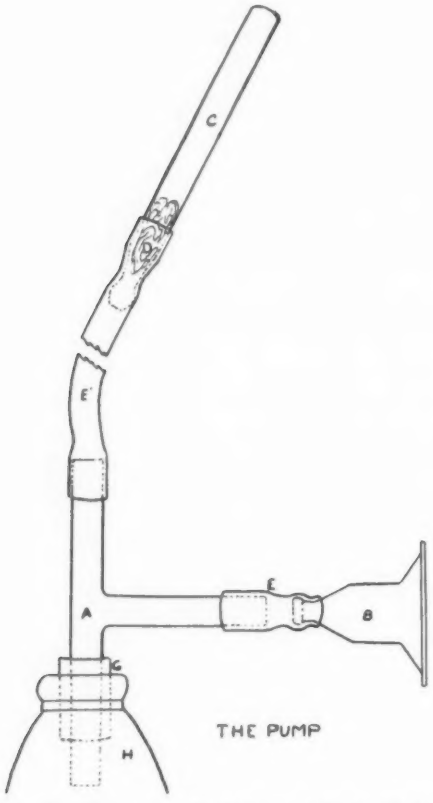

Fig. 1.—Diagram of pump. *A*, T Tube, ⅜ inch tubing; *B*, glass nipple shield; *C*, suction tip; *D*, cotton plug of sterile absorbent cotton; *E* and *E'*, rubber tubing connections; *G*, stopper of rubber tubing; *H*, nursing bottle.

quarter of an inch beyond the edge of the constriction and make a similar constriction. Repeat the process on the other side of the first constriction. Allow the glass to cool and then file on both sides of the central constriction and break the glass at the file marks, leaving a little of the slanting portion so that the end may be easily pushed into the rubber tubing. Round all of the ends by heating them to redness in

the flame. The constrictions thus made serve to hold the cotton in place and to hold the suction tip firmly in the rubber tubing.

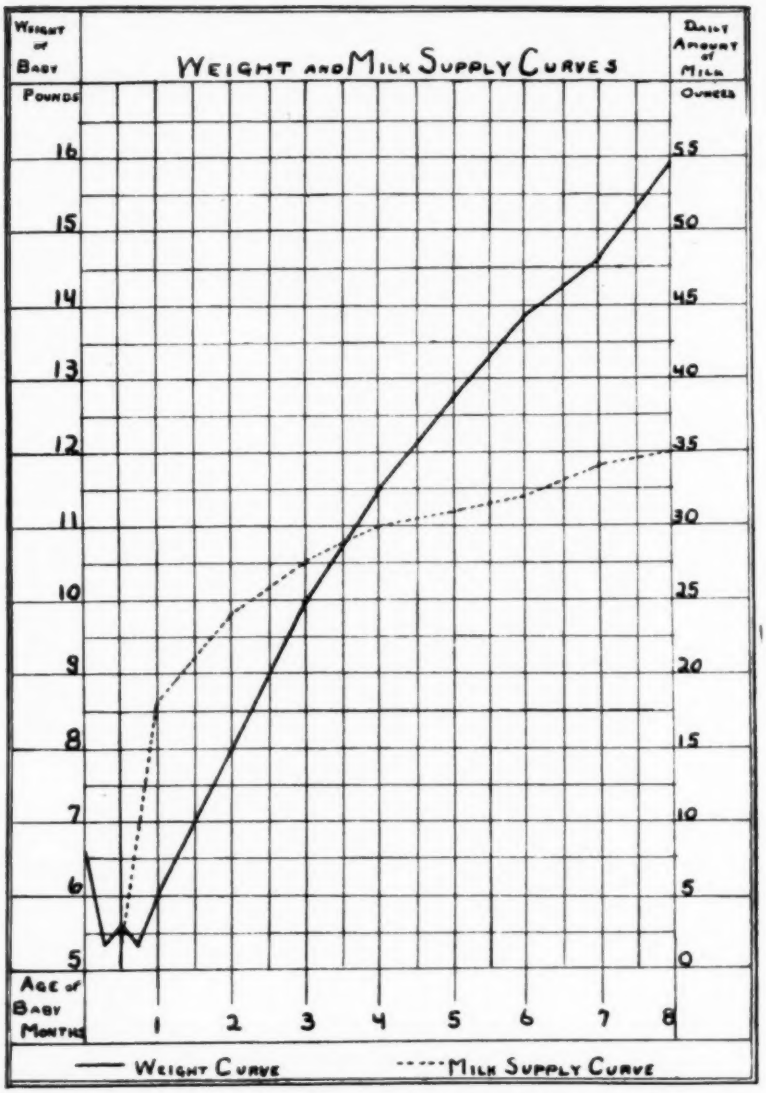

Fig. 2.—Curves of weight of baby and supply of milk, showing increase with use of pump.

4. Non-collapsible rubber tubing of a size that will go over the three-eighths inch tubing and yet will be small enough to make a tight joint with the nipple shield.

5. If the pump is to be used for a considerable time it will be well to have a supply of all parts on hand.

THE ASSEMBLING OF THE PUMP

Though the pump is very simple in construction mistakes can be easily made in putting it together if one is not careful.

1. Join the nipple shield to the side arm of the T tube with a 1¼ inch piece of rubber tubing. (A piece just long enough to join them conveniently.)

2. Fit a 7½ inch piece of tubing over one end of the straight part of the T tube and then put a suction tip into the other end of the rubber tubing.

INCREASE IN MILK SUPPLY AND BABY'S GAIN IN WEIGHT WITH USE OF BREAST PUMP

Date	Weight of Baby		Daily Amount of Milk	
	Pounds	Ounces	Ounces	Grams
February 26...	6	9.5	Apparently	plentiful
March 5	5	7	Less	plentiful
March 12	5	11	10	300
March 19*....	5	7	2	60
March 26†....	6	..	18	240
April 26.....	8	..	24	720
May 26.....	10	..	27.5	825
June 26	11	8	30	900
July 26	12	12	31	930
August 26.....	13	14	32	960
September 26..	14	10	34	1,020
October 26.....	15	13	35	1,050

* Before using pump. † Pump in use.

3. Make a stopper to fit over the free end of the T tube which will fit the mouth of the nursing bottle. This can be done by pushing a piece of rubber tubing (about 1½ inches) over the end of the T tube and then folding the lower end of the rubber tubing over the upper. It may be necessary to slip a second piece of rubber tubing over the first. The nursing bottles and also the T tubes vary in size and the size of the stopper must be changed for each new combination.

4. See that all connections are air-tight by placing the nipple shield against the cheek or hand and making suction through the suction tip.

CLEANING THE PUMP

1. Before using the pump take it apart and thoroughly clean all parts with soap and water, using brushes. Rinse with clear water. Put the pump together and boil it for five minutes. At the same time the bottle and nipple can be cleansed and boiled.

2. After boiling remove both bottle and pump from the water and without touching the lower part of the stopper or the end of the T tube push the stoppered end of the T tube firmly into the mouth of the nursing bottle.

3. Put a plug of sterile absorbent cotton into the suction tip to prevent saliva from running down into the tubing.

Fig. 3.—Pump in use.

TECHNIC OF PUMPING

Place the suction tip in the mouth. With one hand hold the bottle so that the nipple shield comfortably fits over the nipple. With the other hand massage the breast toward the nipple. At first the mouth will become very tired from the sucking, but one soon grows accustomed to making the necessary suction.

RESULTS

1. By means of this pump alone the mother for whom it was made, nursed her baby for over nine months.

2. The mother's breasts never showed any ill effects.

3. The supply of milk increased gradually month by month from 2 ounces to 35 ounces a day.

4. The baby gained 12 pounds and 11 ounces during the nine months, though we had nearly lost hope when we began to use this pump.

5. At the present time the pump has been successfully used by wet nurses at the Presbyterian Hospital of Chicago. In one case the supply of breast milk increased from 18 to 70 ounces per day and then dropped to an average of 54 ounces a day at the end of five months when the woman went home. The one who is there now, obtains an average of 60 ounces a day. I feel certain that by means of this pump any nursing mother can keep up the supply of milk if she is willing to try.

ADVANTAGES OF THE PUMP

1. The pump is effective.

2. It is so simple that any person can make one. Any of the parts can be purchased at a drug store or a laboratory supply store.

3. The pump can be easily cleaned and sterilized.

4. The milk is pumped directly into a sterile bottle, and without contamination can be kept on ice until needed.

5. The exact quantity of milk given the baby is known.

6. The use of the pump does not injure the breasts in any way or cause any soreness.

5205 Greenwood Avenue.

EXPERIENCES WITH THE HIGH CALORIC DIET IN TYPHOID FEVER OF INFANTS AND YOUNG CHILDREN *

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AND

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All pediatrists are familiar with the general course, symptoms and complications of typhoid fever in infants and children when the diet has been limited in accordance with the older ideas of the effect of fever on the digestive capacity. In this society studies of typhoid have been recently reported by Drs. Hand and Gittings, by Drs. Koplik and Heiman and by Dr. Griffith.

One of the main features of typhoid fever has always been the marked emaciation, and closely related to the great loss in weight were the lowered resistance to complications and to relapses and the tendency to toxemic nervous symptoms.

The loss of weight in typhoid, as in starvation and in other fevers obeys the general law that the carbohydrate glycogen stored in the body is at first utilized, and after this the fat and the protein. A characteristic feature of the metabolism of prolonged high fever is the marked loss of body nitrogen.

The loss of weight in typhoid has been attributed to (1) partial starvation, (2) the febrile temperature, and (3) the toxic destruction of protein.

Partial starvation has always been recognized, and the effort has been made by most clinicians to prevent the loss in weight by increasing the diet.

Febrile temperature or artificial heat increases protein metabolism, and probably also the metabolism of fat and carbohydrate. To have such an effect the fever must be high and prolonged. The infectious fevers cause a greater loss of nitrogen than starvation or artificial temperatures, so that it has been assumed that the bacterial toxins exert a destructive influence on the body cells. It has been proved by Coleman and his co-workers that in adults it is possible by the high

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* Read at the Annual Meeting of the American Pediatric Society, Stockbridge, Mass., May, 1914.

* From the Pediatric Service, First Medical Division, Bellevue Hospital, New York.

caloric diet in typhoid to prevent not only the loss of nitrogen, but the loss of weight as well. Weight equilibrium can be maintained in typhoid patients by a sufficient diet.

The main objections to such a diet are: (1) the patients cannot digest and absorb so much food; (2) the amount of fat will cause alimentary disturbance and acidosis; (3) granted absorption, patients do not require the amount of food advocated.

Du Bois has shown that the typhoid patient has a remarkable capacity to absorb large amounts of food. He concludes from his metabolism studies that typhoid patients, throughout the disease, can absorb carbohydrate and protein as well as normal individuals; they can absorb also very large amounts of fat; but the percentage of

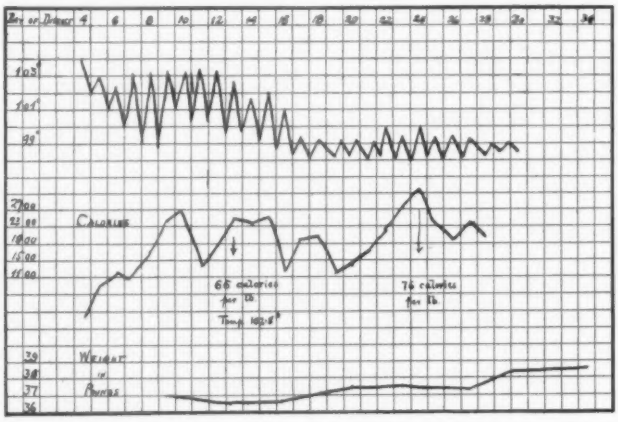

Fig. 1.—High caloric diet in febrile period.

absorption is somewhat lower than the normal, especially in the early part of the disease.

Coleman states that in his 111 cases the increased amount of fat seldom caused any diarrhea, and there was no clinical evidence of acidosis; moreover, no acetone bodies appeared in the urine and the ammonia nitrogen in the urine never exceeded 2 gm., being usually below 1 gm.

As to the food required, it was found clinically that patients lost weight when an excess of food was not given; although an excess of from 1,000 to 2,000 calories were furnished over what the patient expended, there was a loss both of weight and of nitrogen unless such excess was given. In the later stages of the disease the excess is utilized for storing fat.

From the bacteriologic point of view Kendall's[1] studies are important in this connection. He says:

Physiologists have long known that carbohydrate spares body nitrogen, therefore physiological loss of weight due to simple starvation should be largely prevented by such foods. In Coleman's cases not only was the loss of weight attributable to partial starvation prevented, but in addition, practically all the loss attributable to the febrile reaction and to toxemia was markedly reduced. The general symptoms of toxemia were greatly modified.

Kendall further says:

The explanation that presents itself to account for this reduction in toxemia when the patient is on a liberal diet containing an excess of carbohydrates is a simple one. The maintenance of a high physiological sugar content in the body, and consequently in the blood, influences the metabolism of the typhoid

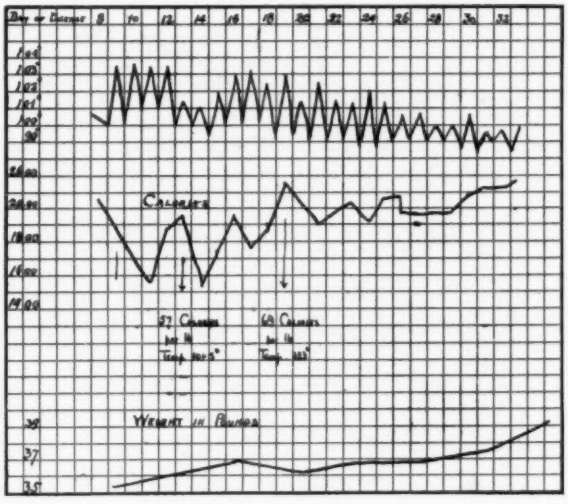

Fig. 2.—High caloric diet in febrile period.

bacilli, directly limiting their action on the body tissues. The typhoid bacilli get their *fuel energy* largely from sugar (dextrose), although their nitrogen requirements are necessarily of protein origin. The waste from the utilization of carbohydrate may be irritative, but is practically never toxic. It is almost certain that the waste products of their *structural* requirements are of comparatively little magnitude. The bacterial waste from fuel needs, particularly when this fuel waste is derived from protein break-down (body tissue in this case) is the more dangerous factor.

It is comparatively simple to understand the changes in the metabolism of the bacteria in the intestinal tract where they are directly in the path of the carbohydrate that is added to the diet and in this connection, the change in the nature of metabolism of the normal endogenous intestinal flora may well play a part. Bacillus coli, for example, forms lactic acid in place of indol, if sugars are available. This may lighten the load on the liver and kidneys, since practically no indol is formed to be excreted through the urinary tract. In the body

1. Kendall, Arthur I.: Boston Med. and Surg. Jour., June 5, 1913, p. 825.

itself, on the contrary, the typhoid bacilli might at first sight, seem to be widely separated from utilizable carbohydrate. Automatically, typhoid bacilli cause focal necrosis in the various organs of the body, but it should be remembered that necrotic tissue is readily permeable to crystalloids. It is very probable that the dextrose of the blood, which is maintained at its highest physiological level on a diet rich in carbohydrate (carbohydrate absorption is only slightly impaired in typhoid) seeps into the areas where the organisms are active and therefore reaches them without difficulty. This dextrose is utilized by the typhoid bacilli in preference to the tissue of their host for their fuel. The character of the products of fuel wastes consequently becomes *acid* in place of being alkaline, nitrogenous and toxic, as would be the case of the dextrose content of the blood were diminished. This explanation involves no theoretical difficulties, and the observed clinical results of a liberal carbohydrate diet in reducing the loss of body protein (loss of weight) in suppressing the toxic state, and in general favorably influencing the course of the disease are in accord with the chemical and clinical results observed empirically in many

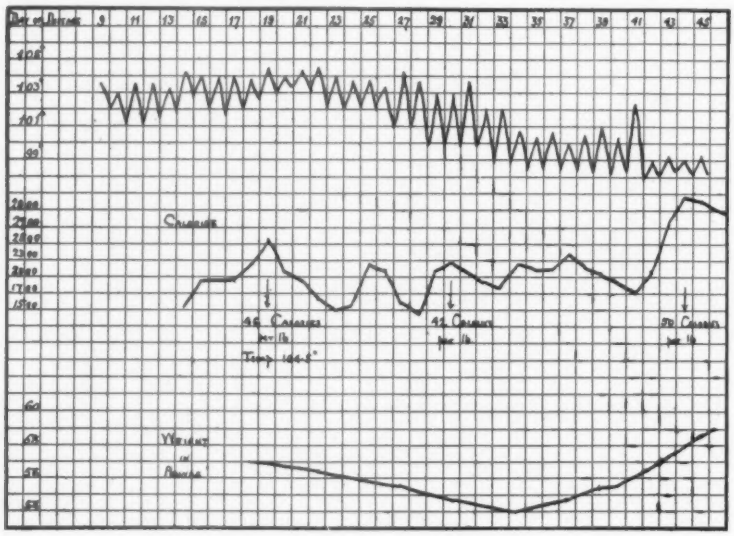

Fig. 3.—High caloric diet in febrile period.

cases where this diet has been introduced. . . . A mortality of 12 among 174 cases of typhoid treated along these lines by Coleman, Croker and Gardner shows what can be done.

Small feedings frequently repeated are important in treating typhoid fever from the bacteriological point of view, so as to maintain a maximal concentration of utilizable sugar, in the intestinal tract and in the body as well. Infrequent feedings fail to do this because the carbohydrate is as a rule rapidly absorbed from the intestinal tract, leaving a residue of protein if the bowel movements are not frequent. The abstraction of the carbohydrate forces the bacteria to utilize protein, be it body tissue or intestinal residuum.

The object of a diet rich in carbohydrate is two fold: *Physiologically* to provide the patient with a readily assimilable food requiring a minimal amount of digestive energy to prepare it for the tissue needs; and *bacteriologically* to shift bacterial metabolism from the destruction of body tissue for their food requirements to the utilization of carbohydrate for at least the major part of their dietary needs. This does not result in annihilation of the invading bacteria, but it certainly approaches their metabolic reformation.

From the point of view of the clinical pathologist, Dr. J. C. Torrey[2] in a recent communication to the New York Academy of Medicine, stated that the high carbohydrate diet encourages the growth of the acidophils; that on a protein diet, such as milk, the putrefactive type predominated; both he and Dr. Charles Norris, the pathologist of Bellevue Hospital, showed that when acidophil bacteria were present in abundance, it was extremely difficult to recover typhoid bacilli from the stools. High fat diet temporarily restrained the growth of acidophils; but when the carbohydrate was resumed, the acidophils grew rapidly again.

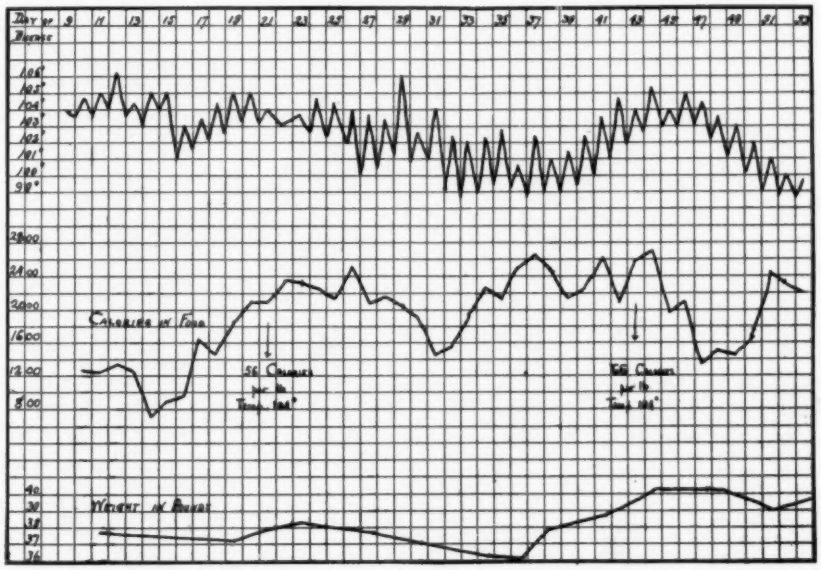

Fig. 4.—High caloric diet. Case with recrudescence.

The metabolism studies of Coleman and Schaeffer and of Du Bois, and the clinical observations of Coleman and his coworkers at Bellevue Hospital, have shown that in adults a much more liberal diet than formerly used is advisable. It was because of interest aroused by this work that we have been anxious to test the value of the high caloric feeding in the typhoid fever of children.

It is seldom that one has the opportunity in New York to see more than an occasional sporadic case of typhoid in children, so that when a considerable epidemic of typhoid occurred in one part of New York, the opportunity was afforded to make a thorough trial of Dr. Coleman's

2. Dr. Torrey's studies have been published under the title, "The Fecal Flora of Typhoid Fever, and Its Reaction to Various Diets," in The Journal of Infectious Diseases, 1915, xvi, 72.

plan of feeding. Some results of our experiences with this diet seem worthy of record. For aid and counsel and for valuable suggestions throughout the epidemic, as well as permission to make free quotations from his writings, we wish to thank Dr. Coleman most heartily.

TREATMENT

The routine treatment was sponge baths for temperature of 103.5 F. The patients were kept on the balconies or in rooms with open windows.

Incidentally it may be stated that typhoid vaccine was administered in seven cases, from one to six doses being used, starting with 50 million and increasing by 50 million each successive dose. There was no perceptible influence on the course of the disease.

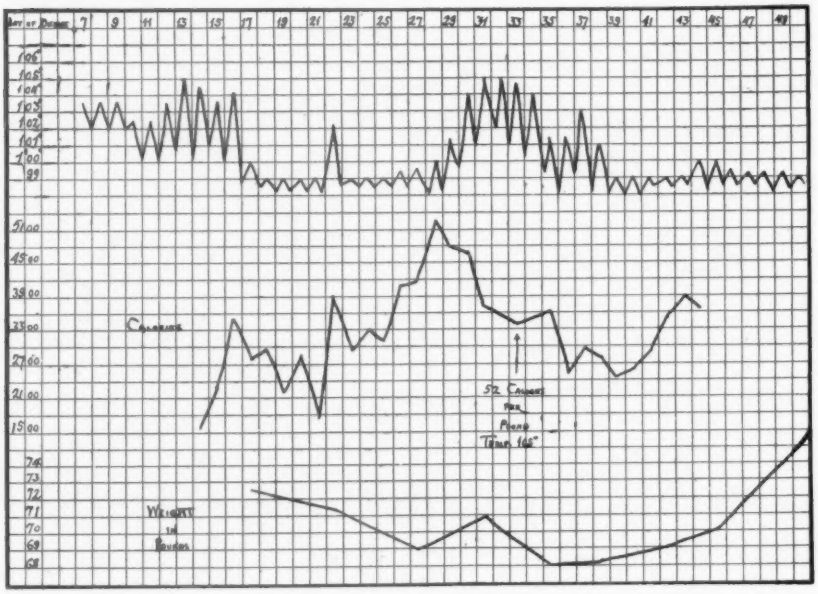

Fig. 5.—High caloric diet in typhoid. Case with relapse.

GENERAL DIET

The children were fed at three-hour intervals, beginning at 6 a. m. and continuing until 9 p. m., six feedings in the twenty-four hours. The articles given were such as would be prescribed for a child in health, with the exception that the only meat used was creamed chicken. In order to increase the caloric value of the liquid food, which was practically always milk, lactose and cocoa and usually cream were added. In no case was an exclusive milk diet used; every patient took cereals in some form. Eggs and toast were offered from the beginning, and it is largely a matter of the nurse's tact and encouragement how

soon a diet high in calories will be taken. At first only a part of the food offered will be taken, and the patient's likes and dislikes must be respected and taken advantage of.

Aside from giving a total amount of food with sufficient caloric value, there must be a certain minimum of protein in the diet. Voit's standard for adults was something over 100 gm. per day, but more recent work has shown that even less would do, but that some 70 gm. should be afforded. Inasmuch as no metabolic studies on children suffering from typhoid have been made, we know of no standard for the daily protein ration in childhood. In our series of cases no attempt was made to have either an upper or a lower limit of protein, but the

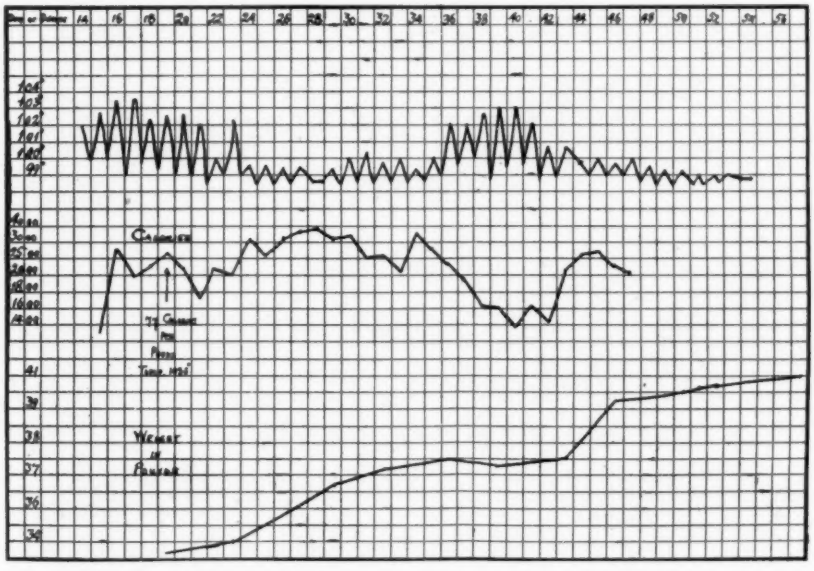

Fig. 6.—High caloric diet in typhoid. Case with relapse.

general rule was adhered to, to have the protein calories not less than 7 nor more than 15 per cent. of the total number of calories given.

Some typical meals of children on the high caloric diet with quantity of food taken and number of calories furnished are given herewith.

TYPICAL DIETS

Angelo R., on the forty-ninth day of disease received 2,214 calories:

6 a. m.: 4 ounces milk, 2 ounces cream, 2 eggs, 2 slices bread.
9 a. m.: 5 ounces milk, 2 ounces cream, 3 crackers.
12 m.: 3 ounces potatoes, 1 egg, 1 slice bread, 6 ounces milk, 2 ounces cream, 2 drams milk sugar.
3 p. m.: 1 ounce ice cream.
6 p. m.: 1 ounce apple sauce, 4 ounces milk, 2 ounces cream, 1 slice bread.

9 p. m.: 4 ounces milk, 2 ounces cream, 2 drams milk sugar.

Leonard B., on November 9 received 2,410 calories:

3 and 6 a. m.: 2 eggnogs with eggs, ½ ounce milk sugar, 2 ounces cream, 4 ounces milk.

6 a.m.: 4 ounces milk.

9 a. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg (eggnog), ½ cracker.

12 m.: Eggnog, 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg.

3 p. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 1 egg.

6 p. m.: Cocoa, 490 c.c.

9 p. m.: Eggnog with 4 ounces milk, 2 ounces cream, 2 eggs, ½ ounce milk sugar.

Leonard B., on December 10, during convalescence, received 7,206 calories:

6 a. m.: 10 ounces cereal, 2 eggs, 4 ounces milk, 2 ounces cream, 4 slices bread.

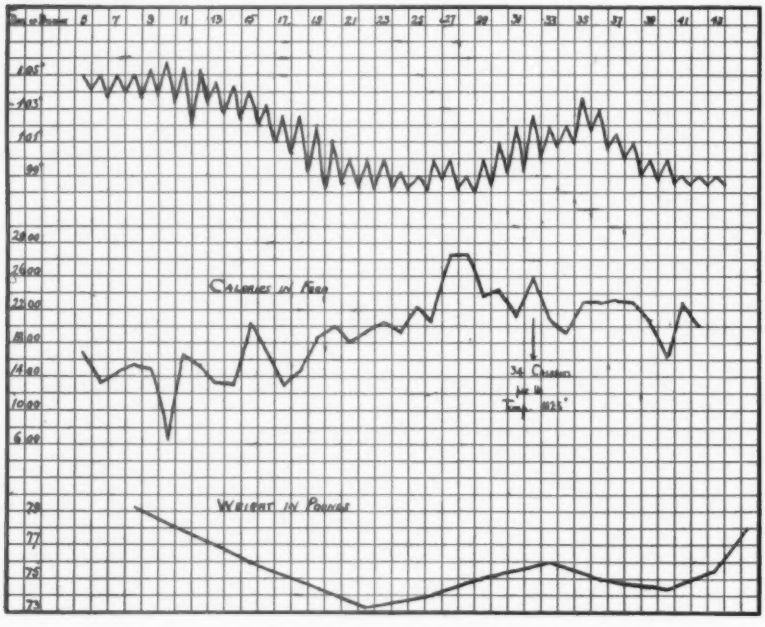

Fig. 7.—High caloric diet. Case with short relapse.

9 a. m.: 4 ounces milk, 2 ounces cream, ½ ounce milk sugar, 4 crackers.

12 m.: 19 ounces potatoes, 6 ounces cream, 4 ounces custard, 8 slices bread.

3 p. m.: 6 ounces milk, 2 ounces cream, 3 crackers.

6 p. m.: 1 egg, 2 ounces apple sauce, 4 slices bread, 4 ounces milk, 2 ounces cream.

9 p. m.: 4 ounces milk, 2 ounces cream, 3 crackers.

J. Corcoran, on the ninth day of disease with temperature ranging from 100 to 103 F., received 2,430 calories:

6 a. m.: 4 ounces milk, 2 ounces cream, ½ slice bread, 1 egg.

9 a. m.: Cocoa, 490 c.c.

12 m.: 4 ounces milk, 2 ounces cream, 1 ounce milk sugar, 1 egg, 1 slice bread.

3 p. m.: 4 ounces milk, 2 ounces cream.
6 p. m.: 4 ounces milk, 2 ounces cream, 1 ounce milk sugar, 1 egg, 1 slice bread.
9 p. m.: 4 ounces milk, 2 ounces cream.

The calculations of the caloric values were made from the following list prepared by Dr. Coleman:

FOOD VALUES — SOLIDS

Food	Amount	Calories
Apple sauce	1 ounce	30
Bread	Average slice (33 gm.)	80
Butter	1 pat (⅓ ounce)	80
Cereal	1 heaping tablespoonful (1½ oz.)	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Custard	4 ounces	250
Cream chicken	1 ounce	50
Egg	1 (2 ounces)	80
Egg (white)	1	30
Egg (yolk)	1	50
Lactose	1 tablespoonful (9 gm.)	36
Milk (whole)	(1 pint 350 gm.) 1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane	1 lump	16
Sugar, milk	1 tablespoonful (9 gm.)	36
Toast	Average slice	80

For practical purposes, the milk sugar may be measured in a medicine glass.

Each measured ounce equals 18 gm. in weight. If milk sugar is added to water in the proportion of 24 gm. to 30 c.c., and the water brought to the boiling point, the milk sugar is completely dissolved. Such a solution made daily, or just before use, will be found convenient in administering the diet.

FOOD VALUES — LIQUIDS

Cocoa with milk:	Calories
1 rounding teaspoonful of cocoa	50
2 ounces of milk sugar	240
4 ounces of milk	80
2 ounces of cream	120
	490

Mix the sugar and cocoa; cook in the milk until dissolved. Serve with cream.

Cocoa with cream:	Calories
1 heaping teaspoonful of cocoa	50
2 ounces of milk sugar	240
½ cup of water	...
3 ounces of cream	180
	470

Mix the cocoa and sugar, add the water and boil. Then add the cream, or use less cream and serve with whipped cream.

Coffee:	Calories
1½ ounces of milk sugar	180
4–5 ounces of strong coffee	...
2 ounces of cream	120
	300

Lemonade:	Calories
4 ounces of milk sugar	480
7 ounces of cold water	...
2 tablespoonfuls of lemon juice (or to taste)	...

Boil the sugar and water for two minutes, add lemon juice to taste, strain and cool.

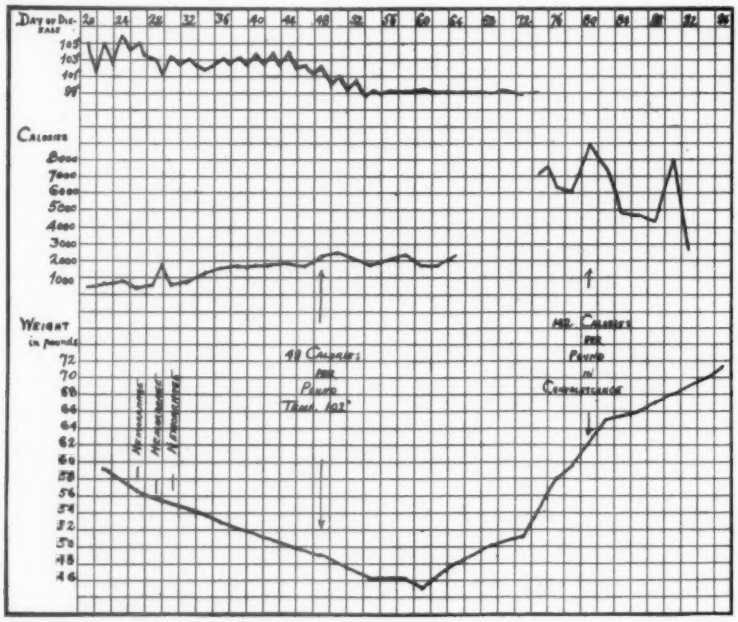

Fig. 8.—Severe case with hemorrhages. Shows loss of weight in spite of liberal feeding during febrile period, but rapid gain in convalescence while taking food of very high caloric value.

The number of calories that were actually taken after the first few days, and that seemed from a clinical point of view to have been satisfactorily utilized, varied from 100 to 300 calories per kilogram. As regards utilization of the food the urine in these cases very seldom showed any more than a slight trace of indican.

It was at first thought that a high range of temperature would in itself interfere with the taking of so much food; this was found to be partially true, but unless toxic nervous symptoms were present, the fever did not prevent the child from taking a generous diet; as the temperature fell, however, the amount of food taken was greater.

The diagnosis was established by positive Widal reaction in forty-four cases and by positive blood cultures in thirty-six cases. In only

one case was there found neither a positive Widal nor a positive blood culture. A complete history of each patient in this series will be found in Tables 1, 2 and 3.

The age range of the patients was as follows: Under 2 years 1 case; from 2 to 6 years, 21 cases; from 7 to 12 years, 26 cases, making a total of 48 cases.

COURSE OF THE DISEASE

The length of time in the hospital averaged twenty-eight days; extremes from thirteen to seventy-nine days. Hand and Gittings report that the average stay in the hospital in their 145 cases was thirty-one days. The ages in their cases ranged from infancy to 12 years, the greater number of the patients being from 4 to 9 years. The day of the disease, as nearly as could be estimated, on which the temperature became normal was found on the average to be the twenty-eighth. Extremes from fifteen to sixty-eight days.

SYMPTOMATOLOGY

The general condition of the patients was remarkably good. The so-called typhoid state occurred in much smaller proportion than under the old methods of treatment, so that the patients did not present, except in rare instances, the typical appearance of typhoid fever.

Abdominal distention.—This was present in only six cases, and in but one of these was it severe or troublesome.

Hemorrhages.—Five patients had, at some time during the course of the disease, blood in the stools. In only two cases were the hemorrhages severe enough to cause any apprehension. In both, blood transfusion was done by the Lindemann method, a procedure which was developed in our wards, and each time with signal benefit. One of the patients, Leonard B., already referred to, was transfused three different times, the amount of blood given being 160 c.c. paternal, the first time, 260 c.c. paternal the second time, and 280 c.c. paternal the third time. This was the patient who lost the greatest amount of weight and who, during his convalescence, took the largest number of calories we have ever seen recorded, namely, 8,000 in twenty-four hours or 310 calories per kilogram. The reason this enormous amount of food was permitted was because of the boy's constant clamor for a cracker or any form of food to satisfy his gnawing hunger. The eternal call for another cracker induced us to let him eat his fill for a few days, with the result reported. He gained in weight from 45 to 72 pounds in thirty-seven days, an average gain per day of about ¾ of a pound.

Nervous Symptoms.—Thirteen patients were irrational in the early stage of the disease. One who came in during the latter part of the second week of his illness presented the picture of a severe psychosis, a type of nervous disturbance occasionally seen late in the course or

TABLE 1.—GENERAL HISTORY OF PATIENTS IN CASES 1 to 43

Case No.	Name	Age, Years	Prodromal Period, Days	Time in Hospital,* Days	Chief Complaint	Nose Bleed	Rose Spots	Spleen Enlargement	Bowels
1	S. Silverman	1 1/12	3	32	Fever	None	++	None	Diarrhea.
2	Freda Gerke	2	10	28	Fever	None	++	None	Regular
3	G. Geiche	3½	7	23	Headache; tire; fever; pain in stomach	None	Appeared in 4 days ++++	+	Loose.
4	A. Dunn	4	2	27	Headache; pain in abdomen	None	++	++	No diarrhea.
5	Margaret Kronek	4	7	14	Fever; headache	?	None	None	Costive.
6	N. Woehinsky	4	7	20	Abdominal pain; headache	None	++	++	No diarrhea.
7	W. Guthart	4½	13	16	Malaise	Yes	++	++	Costive.
8	Sadie Penna	4½	14	26	Abdominal pain; fever	None	++	++	Costive.
9	H. Autmajor	5	3†	4	Fever	None	?	None	Costive.
10	Joseph Cochrane	5	7	28	General malaise	None	None	?	No diarrhea.
11	Catherine Shea	5	6	19	Headache; fever	None	None	+	Costive.
12	M. Geiche	5	5‡	25	Headache; fever; pain in stomach	Negative	+++	+	Loose.
13	R. Zingales	5	5	31	Malaise; headache; fever	None	++	None	None.
14	Rosee Zingales	5	4	21	Headache; fever	None	?	None	Slight diarrhea.
15	George Zoul	5	8	22	"Pain in belly"	None	+	None	Diarrhea.
16	Rocco Garibalii	6	3	18	Fever; headache	None	++	None	Slight diarrhea.
17	Aaron Psau	6	8	16	Fever; headache	?	None	+++	Costive.
18	E. Corcoran	6	7	19	Pain in right side	None	None	None	No diarrhea.
19	Jas. Sloan	6	3¶	43	Fever; occipital; sudden onset; three days	?	None	++	Costive.
20	Catherine Dorso	6½	4	14	Fever	None	++	+++	Costive.
21	John Kelleper	7	8 In bed 2	47	Fever; sleepiness; pain in stomach	?	None	++	Diarrhea for one day.
22	Mike Forlenza	7	14	28	Headache	None	Few ?	None	Constipated.
23	Mary Maucum	8	9	52	Fever; headache	None	++	++	Constipated.
24	Thos. O'Brien	8	7 Bed at once	15	Headache; fever	+ 5 days after adm.	? None	None	Constipated.
25	Coro Guselau	8	5	17	Headache; pain in stomach	?	+++	++	?
26	Olga Medvill	8	3	23	Headache; fever	None	+++	++	More open than usual.
27	Mary Grossman	8	14	44	Headache	None	+++	++	Costive.
28	Julie Schluppe	8	10	28	Headache	Slight	++	++	Loose.
29	Josephine Zanger	9	30	30	Fever	+	++	?	Costive.
30	Josie Campioni	9	7	27	Vomiting; headache	?	None	None	Constipated.
31	Mary Piazza	9	5	34	Fever; headache	None	++	++	None.
32	Angelo Rone	9	10§	..	Fever	None	++	++	?
33	Abe Psau	9	10	16	Headache; fever	None	?	+	Costive.
34	John Purcell	9	10	23	Fever; loss of appetite	None	++	+	Diarrhea.
35	A. Damprosio	10	7	13	Fever; pain in abdomen; headache	Negative	+++	+	Costive.
36	Mary McKenna	10	6	17	Fever; apathetic	None	+++	++	No diarrhea.
37	W. Medville	10	7	31	Chilliness; fever	None	++	++	Costive.
38	Angelina Garibalii	10	5	22	Fever; headache	None	++	++	No diarrhea.
39	C. McCarthy	10¾	7	26	Tire; fever; pain in neck for two days but no headache	Yes	+	+	Costive.
40	F. Windberg	11	6	43	Loss of strength and appetite; fever and slight cough	None	?	+	No diarrhea.
41	Sophie Girdansky	11	3	44	Headache	Slight on day of admiss.	++	None Later ?	Constipated.
42	Rose Newman	11	7	42	Headache; general malaise	None	++	++	
43	Leonard Bisniginns	11	19‖	79	Fever	None	++	Not felt	No diarrhea.

* There is some question as to the length of convalescence.
† Cough 1½ months. ‡ Confined to bed at once. ¶ Sudden onset. § Three weeks before admission. ‖ Sick before admission.

‡ Sudden onset. § Three weeks before admission. ‖ Sick before admission.

TABLE 2.—Tests Made and Complications Seen in Cases 1 to 43

Case No. and Distention	Hemorrhages	Blood Counts	Widal and Blood Culture	Urine	Other Complications
1—None	None	9/7: 11,500 W. C.; P. 42; Ly., 58. 9/10: 17,500 W. C.; P., 57; Ly., 33; L. mono., 6; Trans., 2; Baso., 2.	9/10: W., paratyphoid, pos.	9/7: Albumin light cloud. Sugar, 0.	Both great toes and fourth and fifth fingers of left hand have pus. Double otitis. Gastro-enteritis. Pus shows B. paratyphoid.
2—None; held tense.	None	9/25: 12,500 W. C.; P., 50; Ly., 50.	9/23: W. pos.; 9/27: B. c., pos.	None	No vomiting.
3—None	None	9/13: 17,000 W. C.; P., 60; Ly., 40. 9/22: 10,000 W. C.; P., 34; Ly., 61.	9/18: W., pos. 9/20: B. c., pos.	.....	Refused part of feeding on first four days. Not afterward. Vomited, 16, 17, 19, 21.
4—Slight for two weeks.	None	9/22: 15,500 W. C.; P., 55; Ly., 45.	9/18: W., pos. 9/20: B. c., pos.	9/23: Ft. trace alb. Sugar, 0. Few leukocytes.	Slept fairly well during vomiting period. No mental disturbance.
5—None	None	.....	10/3/13: Widal neg.....	.....	None.
6—Slight on admission and for ten days; then soft.	None. Streak of blood on Octoer 3.	9/16: 12,200 W. C.; Ly., 76; P., 15; L. mono., 9.	9/20: W. pos. 9/22: B. c., pos.	9/23: Sugar, 0. Albumin, 0.	None.
7—None	None	10/14: 9,200 W. C.; P., 43; Ly., 57.	10/13: W. pos. 10/18: B. c., neg.	None	None.
8—None	None	9/26: 5,000 W. C.; P., 58; Ly., 42.	9/27: W. pos. 10/1: B. c., pos.	None	None.
9—None	None	10/9: 6,200 W. C.; P., 50; Ly., 50.	10/9: W., pos.....	.....	Bronchitis? Measles. By Roentgen ray miliary nodules found.
10—None	None	9/30: 10,000 W. C.; P., 66; Ly., 34.	10/2: W., pos. 10/4: B. c., neg.	None	Stye on left eye. Otherwise none.
11—None	None	10/9: 13,400 W. C.; Ly., 20; P., 80.	4/10: W. pos. 6/10: B. c., neg.	None	Vomited three days. October 3, 4, 6.
12—Slight, on admission full through course. Never rigid.	Slight trace of blood on December 2. No change of diet.	9/7: 23,000 W. C.; P., 66; Ly., 34. 9/16: 15,500 W. C.; P., 60; Ly., 59; Baso., 1.	9/18: W., pos. 9/20: B. c., neg.	9/20: Albumin, 0. Sugar, 0. Moderate No. of leukocytes.	Vomited twice. Otherwise none.
13—None	None	18,000 W. C.; P., 67; Ly., 33.	9/23: W., pos. 9/27: B. c., pos.	None	Ecchymosis over left eye. None.
14—None	None	10/28: 9,500 W. C.; P., 56; Ly., 44.	10/29: W., pos. 10:29: B. c., pos.	1/11, 1/12, 1/14: Albumin, neg. Casts, neg. Sugar, neg. Ind., neg.	Readmission; appetite poor until October 9. Then very good.
15—9/19, none; 9/20, slight; 9/25, none.	None	9/19: 23,400 W. C.; P., 77; Ly., 15; L. mono., 8. 9/23: 11,500 W. C.; P., 63; Ly., 37.	9/22: W., pos. 9/25: B. c., neg.	9/24: Albumin, faint trace. Sugar, 0. Casts, 0.	Few crackling râles noted September 20 and September 22 at left apex and right base. Cleared up before discharge.
16—None	None	10/15: 4,000 W. C.; P., 71. Ly., 29.	10/16: W., pos. 10/18: B. c., pos.	None	No nervous symptoms at any time.
17—None	None	9/20: 6,600 W. C.; P., 56; Ly., 42; L. mono., 2.	9/22: W., pos.; 9/25: B. c., neg.	Albumin, trace. Sugar, 0. Few hyaline casts.	
18—None	None	10/9: 6,000 W. C.; P., 75; Ly., 25.	10/10: W., neg. 10/14: B. c., pos.	None	None.

TABLE 2.—Tests Made and Complications Seen in Cases 1 to 43—(*Continued*)

Case No. and Distention	Hemorrhages	Blood Counts	Widal and Blood Culture	Urine	Other Complications
19—None	Slight trace of blood.	8/26: 12,500 W. C.; P., 72; Ly., 28. 8/27: 7,800 W. C.; P., 63; Ly., 30. 9/12: 17,000 W. C.; P., 54; Ly., 46; L. mono., 5.3; Trans., 1.7.	8/17: W., neg. 9/3: W., pos. 9/11: B. c., pos.	8/25: Faint trace albumin. Mammoth pus-cells. 8/26: Albumin, trace. (Hyaline and granular casts many.)	Tenderness and resistance in right upper quadrant.
20—None	None	9/27: 9,500 W. C.; P., 55; Ly., 45.	10/2: W., pos. 10/4: B. c., neg.		None. Uneventful recovery.
21—Slight on admission; none after.	None	9/5: 7,400 W. C.; P., 34; Ly., 54; L. mono., 10; Eo., 2. 8/26: 9,100 W. C.; P., 75; Ly., 13; L. mono., 8; Trans., 4.	8/27: W., neg. 9/27: B. c., pos.	8/26 and 9/24: No sugar. No albumin.	None.
22—Tense for first six days. Then soft.	None	10/29: 10,000 W. C.; P., 57; Ly., 43.	10/29: W., pos. 11/1: B. c., neg.	11/11 and 11/12: Albumin, neg. Sugar, neg. Ind., neg. Casts, neg.	Acute bronchitis for from four to five days.
23—On admission and for sixteen days.	None	9/18: 7,000 W. C.; Ly., 66; P., 34. 9/22: 7,000 W. C.; Ly., 60; P., 40. 10/15: 7,500 W. C.; P., 54; Ly., 46.	9/10: W., pos. 9/14: B. c., pos.	None	Delirious for first week. Boils, parotitis, bronchopneumonia, double otitis media.
24—None	None	9/24: 7,500 W. C.; P., 43; Ly., 57.	9/22: W., neg. 9/24: W., neg. 9/25: B. c., neg.	Albumin, neg. Sugar, neg.	None.
25—None	None	9/18: 5,200 W. C.; P., 54; Ly., 36; L. mono., 10.	9/22: W., pos. 9/22: B. c., neg.	Albumin, 0. Sugar, 0.	None.
26—None	None	9/9: 10,500 W. C.; P., 60; Ly., 40.	9/14: W., pos. 9/16: B. c., pos.	Sugar, 0. Albumin, 0.	None.
27—Slight; none after relapse.	None	None	9/13: W., pos. 9/14: B. c., pos.	9/22: Sugar, 0. Albumin, 0. Mod. number of leukocytes.	Vomited once on thirty-seventh day.
28—None	None	9/25: 5,500 W. C.; P., 53; Ly., 47.	9/27: W., pos. 10/1: B. c., neg.	None	Bronchitis (slight).
29—Slight at first; none afterward.	Two	9/30: 5,000 W. C.; P., 70; Ly., 30. 10/17: 6,600 W. C.; P., 65; Ly., 35.	10/3: W., pos. B. c., no report.	None	Bronchitis. Hemorrhage. Vomited.
30—None	None	10/6: 10,000 W. C.; P., 54; Ly., 46.	10/8: W., pos. 10/13: B. c., pos.	None	Headache; poor appetite for first nine days. Then better.
31—Some; tense for two weeks.	Trace on 10/3; none after.	9/20: 10,000 W. C.; P., 74; Ly., 26. 10/6: 10,000 W. C.; P., 60; Ly., 40. 10/8: 15,000 W. C.; P., 45; Ly., 55.	10/2: W., pos. 10/4: B. c., pos.	None	
32—Tense	None	11/12: 14,000 W. C.; P., 60; Ly., 33; Trans., 1; L. mono., 6. 11/26: 6,400 W. C.; P., 50; Ly., 50. 11/28: 6,000 W. C.; P., 48; Ly., 52. 12/6: 12,600 W. C.; P., 61; Ly., 39.	11/15: W., pos. 11/19: B. c., pos.	12/12, 12/27: Sugar, neg. Albumin, neg. Ind., neg.	Acute bronchitis on admission; bronchopneumonia, boils; perirectal abscess.

33—None	None	9/20: 9,800 W. C.; P., 49; Ly., 49; L. mono., 2.	9/22: W., pos. B. c., pos. 9/25:	Albumin, marked trace. Few ly., 0. Gr. casts.	Ring worm on scalp. Red papular eruption. Wassermann positive. Father's Wassermann strongly positive.
34—None on admission; held abdomen tense.	None	9/18: 7,600 W. C.; P., 38; Ly., 53; L. mono., 6; Eo., 3.	9/22: W., pos. B. c., pos. 9/25:	9/23: Albumin, faint trace. Sugar, 0. Ca. oxal crystals.	Delirious, very drowsy and apathetic for six or seven days. Then brighter gradually.
35—Moderate at first. Later soft.	None	9/24: 6,500 W. C.; P., 60; Ly., 40. 9/29: 15,000 W. C.; P., 78; Ly., 22.	9/26: W., pos. B. c., pos. 9/28:	9/25: Albumin, faint trace. No sugar. No casts.	Bronchopneumonia with many small abscesses in right lower lobe.
36—None. Slight tenderness over abdomen on admission.	None	9/19: 7,400 W. C.; P., 51; Ly., 41; L. mono., 5.	9/22: W., pos. B. c., pos. 9/25:	9/13: Albumin, very faint trace. Sugar, 0. Sediment, 0.	None.
37—Complained of abd. pain, but not distended. Cleared up quickly.	None	9/13: 9,000 W. C.; P., 71; Ly., 29. 10/14: 7,000 W. C.; P., 25; Ly., 75.	9/14: W., pos. B. c., pos. 9/16:	9/14: Albumin, faint trace. Few leukocytes. Hyaline casts. Amor. urates. Sugar, 0.	Scoliosis. Otherwise none.
38—None	None	10/13: 6,600 W. C.; P., 66; Ly., 34.	10/13: W., pos. B. c., pos. 10/14:	None	Vomited several time. Uneventful recovery.
39—None	None	9/10: 9,000 W. C.; P., 71; Ly., 29.	9/11: W., pos. B. c., neg. 9/14:	Slight trace of albumin.	None.
40—None during relapse.	None	9/22: 9,500 W. C.; Ly., 59; P., 41. 9/24: 12,500 W. C.; P., 48; Ly., 42.	9/3: W., pos. B. c., pos. 9/6:	9/3: Albumin light cloud. Sugar, 0. 9/14: Albumin, 0. Sugar, 0.	None.
41—Full but not distended.	.....	10/18: 11,000 W. C.; P., 50; Ly., 50. 10/31: 9,000 W. C.; P., 47; Ly., 53. 11/21: 9,000 W. C.; P., 51; Ly., 49.	10/18: W., pos. B. c., pos. 10/20:	11/11, 11/12, 11/14, 11/26: Albumin, sugar and indican, neg.	Boil on right thigh. Vomited once before admission.
42—9/21, slight; 9/24, abd. soft. Soft thereafter.	None	.....	9/5: W., pos. B. c., neg. 9/9:	9/24: Albumin, faint trace. Sugar, 0. Triple phos.	Bronchitis for first three weeks. Nose bleed.
43—Moderate on admission.	10/17: slight. 10/18: 3; 10/19: severe; 10/20: 2; 2 sev.; 1 slight. 10/21: blood in stool; 10/22: hemorrhage.	10/13: 19,000 W. C.; P., 51; Ly., 43; L. mono., 5; Baso., 1. 10/19: 6,600 W. C.; P., 60; Ly., 40. 10/22: R. C., 4,800,000; Hb., 58%. 10/24: 16,000 W. C.; P., 24; Ly., 76. 10/26: 9,500 W. C.; P., 44; Ly., 56. 10/28: 8,500 W. C.; P., 40; Ly., 60; R. C., 3,500,000; Hb., 45%. 11/13: 10,000 W. C.; P., 41; Ly., 51; Hb., 53%; R. C., 2,500,00. 11/17: 6,200 W. C.; P., 36; Ly., 70; Hb., 50%; R. C., 3,000,000. 12/10: Hb., 79%; R. C., 5,700,000; W. C., 17,000; P., 41; Ly., 59.	10/18: W., pos. B. c., pos. 10/20:	11/26: Albumin, trace. Sugar, 0. Indican, ?. 12/8: Albumin, trace. Sugar, 0. Indican, trace. 12/26: Albumin, neg. Sugar, neg. Indican, neg. 10/22: Albumin, mod. trace. Few hyaline and granular casts. Few leukocytes.	Acute bronchopneumonia. Lobar. Acute hepatitis? Cholecystitis? Decubitus. Acute myocarditis.

TABLE 3.—RELATION OF FEEDING TO GENERAL COMFORT AND NERVOUS SYMPTOMS

Case No.	Relapses	Condition of Patient	Medicines	Vaccine
1	None.....	Extremely irritable for three weeks. Appetite never good	Hexamethylenamin.....	None.
2	None.....	Extremely irritable during entire stay, but never really sick. Appetite fair.	None.....	None.
3	None.....	Very nervous and irritable for ten days during which appetite was poor, then better. Condition continued better, as did appetite.	Hexamethylenamin last 7 days.	None.
4	None.....	Restless on admission. Mentality not impaired. Apathetic and irritable for first two and one-half weeks.	None.....	None.
5	None.....	Child was in excellent condition from start to finish.....	None.....	10/30, 200 million.
6	None.....	Apathetic first few days. Later good. Appetite good.....	Hexamethylenamin.....	None.
7	None.....	Very irritable and ill on admission. Appetite fairly good. No nervous symptoms.	None.....	None.
8	None.....	Abdomen held tense on admission. Quite restless for first week. Appetite then good.	None.....	None.
9	.....	Had a few râles at bases.....	.....	None.
10	None.....	Appetite poor for nine days. General condition always good. No nervous symptoms.	None.....	None.
11	None.....	Not stuporous or prostrated on admission. Cried a great deal first few days. Appetite poor ten days then good. No nervous symptoms.	None.....	None.
12	None.....	Appetite poor for five days, then better. No mental disturbance after six days.	Hexamethylenamin.....	None.
13	None.....	Abdomen held tense. Appetite poor for eighteen days. Very quiet for first two weeks. No nervous symptoms.	None.....	None.
14	This attack was a relapse	Very quiet but never had nervous symptoms.....	None.....	None.
15	None.....	Appetite very poor until October 3. Very uncomfortable until September 26, when general condition of nervousness became better. Had headache and was restless.	Caff. Sod. Benz.....	None.
16	None.....	Appetite poor first few days; good for two days then poor again. Picked up finally and had good appetite.	None.....	None.
17	None.....	Appetite poor first seven days; then good. Little sleep first two days; then bright.	Hexamethylenamin.....	None.
18	None.....	Appetite poor but general condition good. Appetite better after four days. No nervous symptoms.	None.....	None.
19	None.....	.....	Pot. Cit. Urot. Calc. Lact.	None.
20	None.....	Always in good condition. Apathetic for a day or two, but then better.	None.....	None.
21	None.....	Had good appetite from start. Never very sick.....	Hexamethylenamin.....	None.
22	None.....	Appetite poor for ten days. Very quiet.....	None.....	None.
23	On fortieth day, but no addition nor symptoms.	September 14, pulse imperceptible. September 16, rational for first time. September 18, irrational again. September 19, brighter.	None.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 150 million; 10/2, 200 million.
24	None.....	Appetite and general condition always good.....	None.....	200 million.
25	None.....	Appetite usually poor; no disturbance of mentality. Well at all times.	Hexamethylenamin.....	None.

26	None.....	Apathetic for first few days. Appetite good always.....	Hexamethylenamin.....	None.
27	Relapse, but general condition remained excellent	Appetite good for first ten days; rather poor on September 15 and 16, but nervous condition at all times good. Up in chair September 20 for two hours till September 26; then to bed again. Appetite good.	Hexamethylenamin.....	None.
28	None.....	Appetite poor for first week. Never very sick.....	None.....	None.
29	None.....	Hemorrhage day after admission and two days later. Transfused and horse serum 30 c.c. injected. Father's blood used in transfusion. Restless and irrational till October 5.	9/30, 15 c.c. horse serum; 10/1, 30 c.c. paternal blood; 10/3, 20 c.c. horse serum; Calc. Lact.; hexamethylenamin.	None.
30	None.....	Refused food for first week. No nervous upsets except headache.	None.....	None.
31	None.....	Delirious for nineteen days with marked meningeal symptoms. Double ankle clonus; stiff neck. Spinal cord tap cells 3. Noguchi negative. Appetite improved October 7.	Hexamethylenamin, salol.	None.
32	None.....	Child was admitted in delirium with meningismus, November 19. Temperature dropped from 105 to 97.6 F. For ten days was nervous and very restless.	Hexamethylenamin.....	None.
33	None.....	Stupid for two days but always had a good appetite.....	Hexamethylenamin last 5 days.	None.
34	None.....	Appetite very poor for seven days then gradually improved. Comfortable first on October 2.	Hexamethylenamin.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 100 million; 10/1, 100 million.
35	None.....	Irrational from second day onward.....	.....	9/28, 50 million; 9/30, 150 million; 10/2, 200 million.
36	None.....	Appetite good from start. No nervous disturbance.....	Hexamethylenamin.....	9/11, 50 million; 9/13, 50 million; 9/15, 100 million; 9/17, 100 million; 9/19, 100 million; 9/22, 200 million.
37	None.....	For first two weeks was very apathetic and irrational the first few nights, then better; appetite poor usually; good the last two weeks.	Hexamethylenamin.....	None.
38	None.....	Appetite only fairly good for eleven days, then good. No nervous upsets but cried a great deal on October 14.	None.....	None.
39	None.....	No mental disturbance at any time. Appetite always good	Hexamethylenamin seven days.	None.
40	After three weeks. Made good recovery	Appetite good from start. Only fairly so during relapse for the first three days. Then good again. No nervous symptoms. Somewhat sleepy.	9/17, 9/20, hexamethylenamin.	None.
41	? Had boil.....	Actively delirious on October 24; lasted about one week. Appetite very poor and very weak until October 28; then appetite improved but never very good.	None.....	None.
42	None.....	Appetite rather poor but no nervous disturbances.....	None.....	9/22, 50 million; 9/24, 50 million; 9/26, 100 million; 9/28, 100 million; 9/30, 150 million; 10/2, 200 million.
43	None.....	Child was delirious and weak until early in November. Until November 19, the child did not have one comfortable night; after that slowly improved.	Hexamethylenamin; brandy; Calc. lactate; cod liver oil; wine of iron; Tr. nux vomica.	10/18, 20 c.c. horse serum; 10/19, 160. c.c. paternal blood; 10/20, 260 c.c. paternal blood 7 a. m., 80 c.c. saline intravenously; 10/22, 1 p. m., 30 c.c. saline intravenously; 10/23, 20 c.c. horse serum; 11/13, 280 c.c. paternal blood intravenously.

following the disease under the old methods of treatment. This nervous state was demonstrated to be a starvation psychosis. After a few days of forced feeding the boy came out of his stupor and delirium and became entirely rational. There is not the slightest doubt that most of the severe nervous symptoms noticed in the late stages of the disease heretofore were due to the starvation and the consquent increase in the toxemia.

Complications.—Pneumonia or bronchopneumonia were present in three cases, and in the only death that occurred the fatal issue proved by necropsy to be due to pneumonia.

Relapses.—Among our cases there were four relapses and one recrudescence, or a little over 10 per cent. Koplik and Heiman report 24 relapses among their 160 cases treated at the Mt. Sinai Hospital, a percentage of 15. Hand and Gittings had 4.8 per cent. of relapses in 145 cases.

Mortality.—There was one death, a mortality of about 2 per cent.

During the course of the fever, 60 per cent. of our patients gained in weight. This was the most remarkable fact about this series of cases; and is, in reality, the reason for this report. Heretofore the loss in weight has been very marked throughout the course of the disease, so that by the time the temperature has become normal, the patient has usually presented the picture of emaciation. The high caloric diet prevents this loss in the great majority of cases or reduces it to a very small amount. If we consider the weight of the patient at the time of leaving the hospital, it is found that 92 per cent. had gained in weight.

In 39 cases there was a gain in weight over that on admission as follows:

Pounds	Cases	Pounds	Cases
¼	1	4	1
½	1	4¼	2
¾	1	5	1
1	1	5¼	1
1½	3	6	1
2	4	6½	1
2¼	3	7¼	3
2½	2	7½	2
3	3	9	1
3¼	3	9¼	1
3½	2	11½	1

CONCLUSIONS

1. It is possible, in the great majority of cases, to feed children suffering from typhoid fever, even during the febrile period, with an amount of food furnishing sufficient calories to maintain or even increase their body-weight.

2. This high caloric diet, besides preventing the emaciation usual in this disease, greatly increases the comfort of the patient, prevents the severe nervous symptoms and lessens the dangers of the disease and of its complications.

THE ROENTGEN RAY IN THE DIAGNOSIS OF PULMONARY CONDITIONS IN CHILDREN*

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NEW YORK

Although the importance of the Roentgen ray in the diagnosis of pulmonary tuberculosis has for some time been recognized, it is only within the last year that the possibilities of its use in the diagnosis of other pulmonary conditions has been developed.

Pneumonias showing few physical signs, due to their location and to the slight amount of lung tissue involved, serous and purulent exudates, abscess of the lung, hyperplasia of bronchial lymph-nodes, pneumothorax, as well as miliary tuberculosis, are now visibly demonstrated where formerly their existence was largely a matter of conjecture.

In the children's service at the Roosevelt Hospital, roentgenograms are taken of all pneumonias, not only in obscure cases for diagnosis, but also to determine associated conditions and the extent of active processes.

The cases reported illustrate the value of its aid in these various conditions.

The first roentgenogram (Fig. 1) shows a practically normal chest. The slight shadow at the right and left of the heart is called the hilum shadow and is due to the branching of the bronchi and vessels at the root of the lungs. In some cases the mediastinal glands are distinctly pictured.

In pneumonia the shadow in the lung tissue is produced by the congestion and hepatization there present. Many of these conditions are better shown in the negative than in the prints.

CASE 1.—Pneumonia. Philip O'D., aged 10 years. Admitted August 6, discharged Aug. 20, 1913. Family and past history negative.

Present Illness.—Four days ago complained of headache, referred to the frontal region, pain in the epigastrium and in the lower right chest, worse on inspiration. Has had cough and bloody expectoration. Vomited five or six times; bowels have been loose; anorexia.

Examination of the Lung: Good respiratory excursion, respiratory murmur is accentuated over the right lower chest anteriorly. No adventitious sounds heard.

On admission temperature was 101, repirations 28, pulse 140. Temperature that night rose to 104 and remained at 104 with slight remissions for three days when it fell by crisis to normal.

* Received for publication Feb. 4, 1915.

* From the Bliss Ward, Pediatric Service of the Roosevelt Hospital.

Blood Examination	Aug. 6	Aug. 8
White blood corpuscles	40,000	27,400
Polymorphonuclears	94	87
Lymphocytes	6	8
Hemoglobin	95	

August 8.—Still has harsh and sibilant breathing over right anterior chest, but no definite signs of pneumonia. Roentgenogram (Fig. 2) shows partial consolidation of the right upper lobe, with extensive involvement of the middle lobe.

August 9.—Physical signs show typical bronchial voice and breathing over the right upper and middle lobes.

CASE 2.—Pneumonia.—Harold W., aged 7 months. Admitted July 21, discharged July 31. Family history negative.

Present Illness.—Breast fed for six months, then given milk and barley water. Four days ago began to cough and soon developed a high fever. Had one convulsion lasting twenty minutes, very dyspneic. Another convulsion on the day of admission. Marked rachitis.

Examination of the Lungs: Over the left posterior chest dulness with many coarse râles. On both sides friction rubs are heard. Temperature on admission 102, rising to 104, and ranging between 100 and 103 for three days, falling to normal on the fifth day. Respirations 36 to 80, pulse 130 to 160.

Blood examination:

White blood cells	13,100
Polymorphonuclears	40
Mononuclears	9
Hemoglobin	60

Lumbar puncture: fluid not under pressure:

White blood cells	30 to cm.
Polymorphonuclears	65
Lymphocytes	33
Mononuclears	2

Roentgenogram (Fig. 3) three days after admission showed a pneumonia involving the upper right and left lobes extending to the right middle lobe.

CASE 3.—Pneumonia. Woodford, aged 15 months. Admitted July 28, discharged Aug. 18, 1913. Father died of tuberculosis.

Present Illness.—Breast fed for seven months, then given modified milk and light diet. Three days ago the patient developed fever accompanied by a slight cough. For two days past the child has refused to lie on the right side. No history of chill.

Examination of the lungs: Anteriorly on the right side there is dulness from the supraclavicular space to the base, with bronchial voice and breathing down to the fourth rib. Left side of chest is clear.

Temperature 102, rising to 104. 4 in the afternoon. Respirations 80. Pulse 72. Temperature fell gradually to 100 with slight evening exacerbation. Patient developed a double otitis media and left the hospital against advice.

Two days after admission the Roentgenogram (Fig. 4) showed a pneumonia involving nearly all of the right side.

CASE 4.—Miliary tuberculosis. James S., aged 10 months. Admitted Oct. 14, 1913, died Oct. 15, 1913. Family history negative.

Present Illness.—Patient had been under observation in the outpatient department for over one month, brought first because of a swelling on the left side of the neck and also suffering from an enteritis. At this time the examination of the lungs shows many coarse râles widely distributed. The cervical adenitis responded very slowly to treatment and the child lost weight rapidly. The baby

was taken to the country but failed to improve. He was admitted to the hospital with a temperature of 100, respirations 56, pulse 156.

Examination of the Lungs: Chest normal contour, no rosary present, respiration somewhat limited over the right side and labored. Over the whole of the right chest are heard many mucous râles. There is dulness and marked increase in the voice and breathing in the axilla, extending upward over the middle and upper lobes. Behind there are showers of large moist râles over the entire chest, more concentrated at the apices.

Temperature rose that evening to 102.8, falling in the morning to 98.

Blood examination:

White blood cells	10,700
Polymorphonuclears	75
Lymphocytes	11
Mononuclears	4
Hemoglobin	48

The roentgenogram (Fig. 5) showed both lungs studded with miliary tubercles. After death the peritoneal glands were easily palpable.

CASE 5.—Miliary tuberculosis. Philip M., aged 2 years. Admitted July 19, discharged July 31, 1913. Family history negative.

Present Illness—One month ago had measles followed by chicken-pox which lasted for two weeks, when he developed a high fever and a cough. Has had no night sweats, but has lost considerable weight.

Examination of the Lungs: In front many fine and coarse râles are heard over the entire chest. Behind there is dulness on both sides extending down as far as the spine of the scapula. Over the entire posterior chest are heard many fine râles. There is no change in the voice and breathing.

Temperature ranged from 101 to 104 in the evenings. Respirations 60 to 70. Pulse 160 to 180.

Blood examination:

White blood cells	21,900
Polymorphonuclears	54
Lymphocytes	30
Mononuclears	15
Eosinophils	1
Hemoglobin	78

Von Pirquet positive.

Roentgenogram (Fig. 6) showed many small areas apparently miliary tubercles over both chests.

Condition unimproved at discharge.

CASE 6.—Bronchopneumonia with serous exudate. Alexander M., aged 10 months. Admitted September 24, discharged Oct. 28, 1913. Mother is in the hospital at present with lobar pneumonia. Mother and child were admitted on the same day.

Present Illness.—Three days ago child was taken to a dispensary where a diagnosis of bronchitis was made. Chief complaint has been cough accompanied by high fever.

Examination of the Lungs: Slight dulness of the right base behind. Temperature ranged from 100 to 104 in the afternoons for four days when it fell to normal. Respirations 28 to 60.

Roentgenogram (Fig. 7) showed a bronchopneumonia with exudate involving the right side. A week later there was a reinfection with marked dulness and diminished breathing at the right base, causing a temperature which ranged from 98.8 to 105 for four days.. Complicating this was a double otitis media and a general furunculosis which gradually subsided.

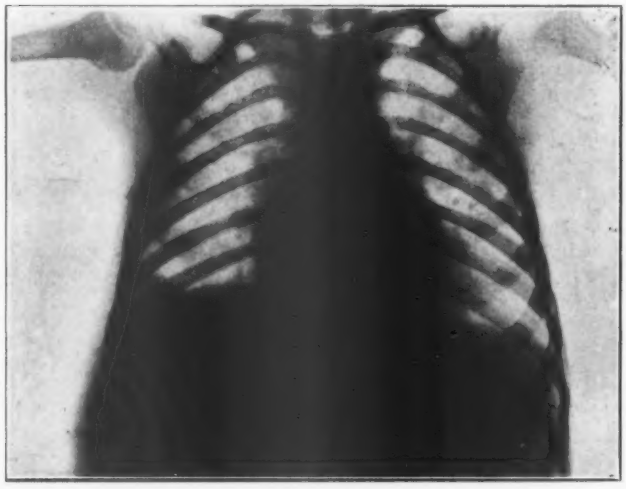

Fig. 1.—Practically normal chest. The slight shadow at the right and left of the heart, the hilum shadow, is due to the branching in the bronchi and the vessels at the root of the lungs.

Fig. 2.—Case 1, pneumonia. The house physician reports that this case was first seen by the surgeons because of the severity of the abdominal pain which radiated to the right lower quadrant. The diagnosis rested between an acute catarrhal appendicitis and a developing pneumonia. The patient was finally referred to the medical service and was of unusual interest as the physical signs did not correspond with the clinical severity of the attack. It was several days after the picture had shown an involvement of the right upper and middle lobes that these signs corresponded with a lesion of such extent.

Fig. 3.—Case 2, pneumonia. When first seen this patient appeared to be suffering from some severe form of cerebral irritation. The child's head was thrown back; the respirations were exceedingly rapid, from 60 to 80 most of the time. Physical signs did not indicate the pneumonic areas involved and a meningeal condition was suspected. Lumbar puncture did not confirm this possibility. A roentgenogram showed distinctly a developing pneumonia, the signs of which soon appeared.

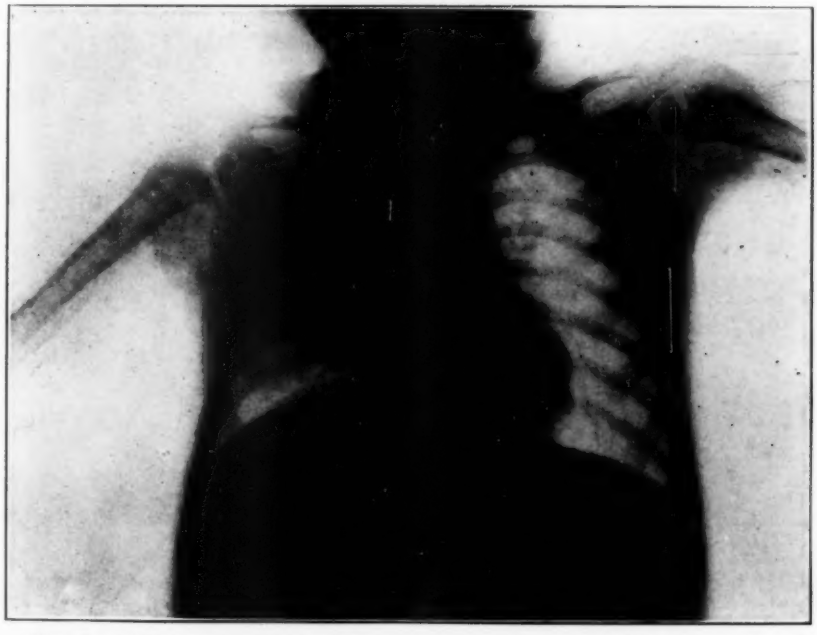

Fig. 4.—Case 3, pneumonia. The extent of the pneumonia is clearly shown. The physical signs at the time the picture was taken were very definite.

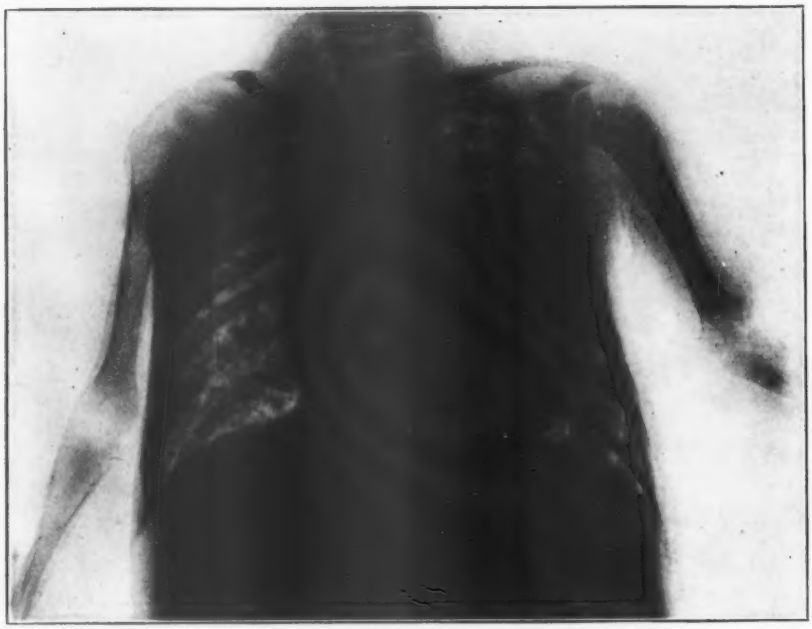

Fig. 5.—Case 4, miliary tuberculosis. Both lungs studded with miliary tubercles.

Fig. 6.—Case 5, miliary tuberculosis. It was thought on admission that the child might be suffering from a diffuse bronchopneumonia. The general course of the disease with the physical signs and the roentgenogram pointed clearly to a pulmonary, and probably a general, miliary tuberculosis.

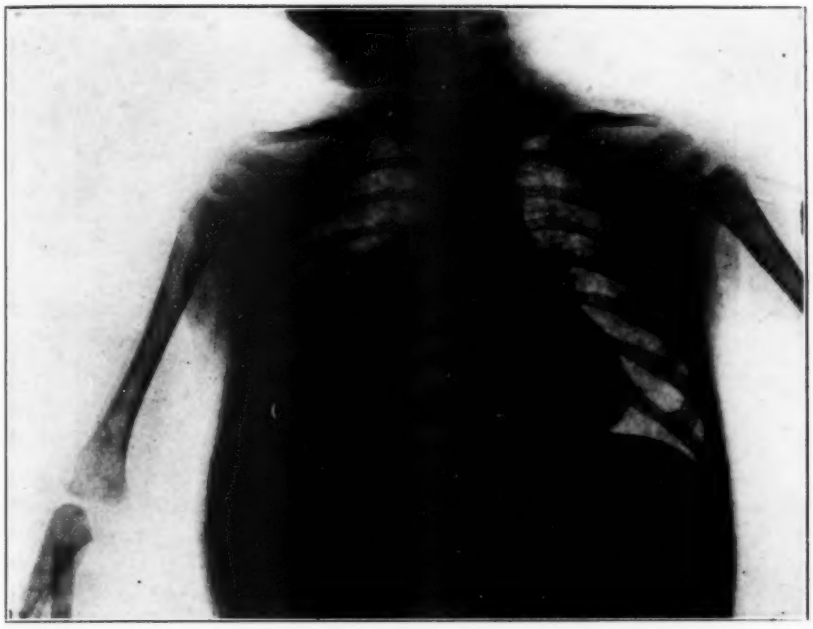

Fig. 7.—Case 6, bronchopneumonia with serous exudate. This picture shows very well the obliteration of the costal-phrenic angle on the right side, indicating the presence of exudate. No free fluid was found on paracentesis of the chest. The physical signs gradually disappeared, so it was concluded that the exudate had been absorbed. The child was discharged in good condition.

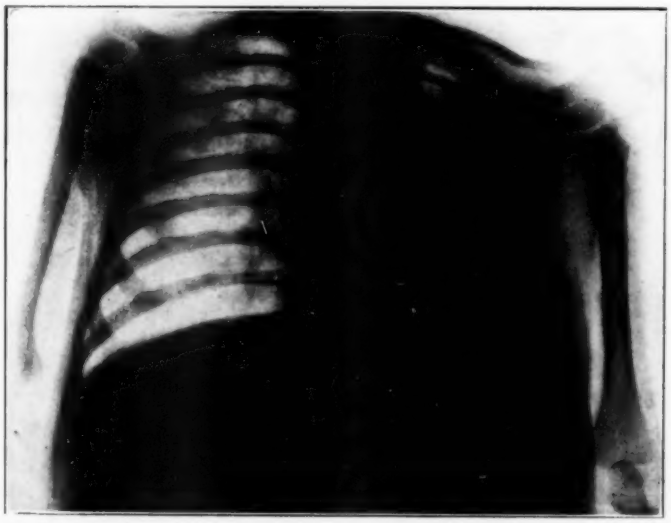

Fig. 8.—Case 7, abscess of the lung. This picture also shows the obliteration of the costal-phrenic angle on the left side. The diagnosis of empyema was made from the general physical signs and from the condition of the child. Although no pus was found at the time of the operation, it was felt certain that the lung was infected and consequently the wound was left open with drainage. The result showed that this conclusion was correct.

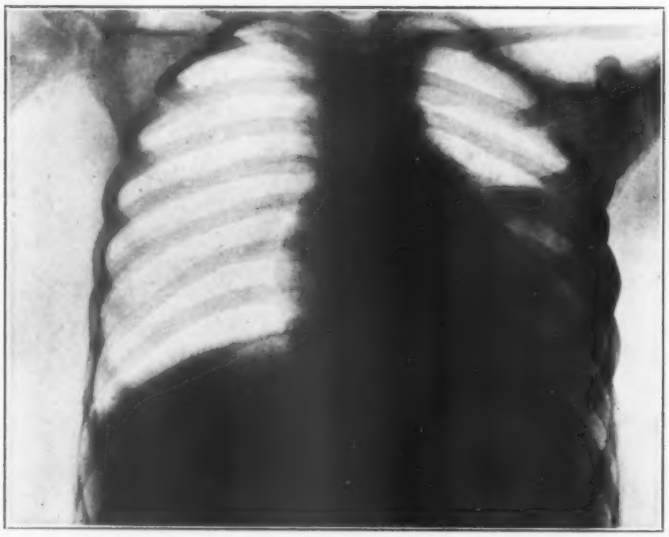

Fig. 9.—Case 8, abscess of the lung. July 12: Distinct shadow extending from the middle of the left chest to the base. There is a darkened area near the top of the shadow.

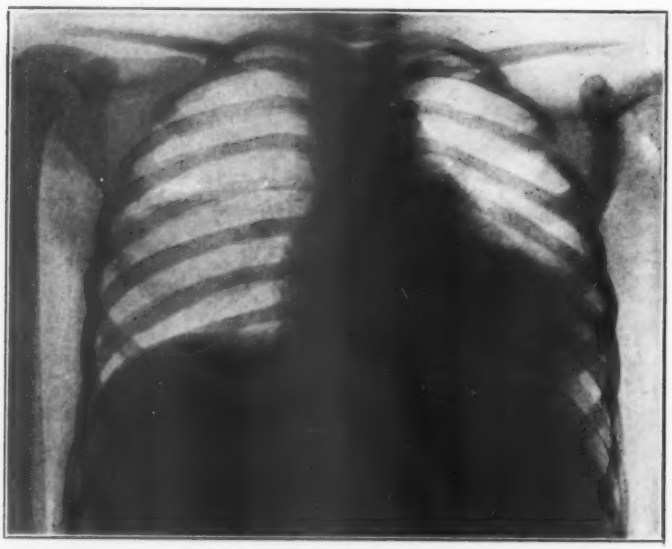

Fig. 10.—Case 8, abscess of the lung. July 22: Condition about the same, physical signs about the same. Area about the size of a dollar well defined.

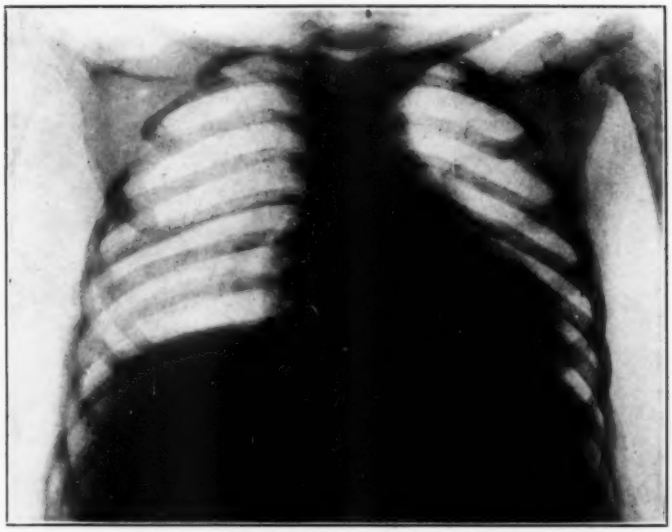

Fig. 11.—Case 8, abscess of the lung. July 31: Area is smaller and less distinct. The signs in the chest have nearly disappeared.

Fig. 12.—Case 9, pyopneumothorax. This shows very well the sacculated area of exudate with air above it. A thick band of adhesions apparently extends upward over the pocket. There is involvement of the lungs on both sides.

Fig. 13.—Case 9, pyopneumothorax. This picture is taken with the patient on her side and shows the change in the position of the air and exudate.

Fig. 14.—Case 9, pyopneumothorax. In this picture the sinus leading to the pocket is injected with bismuth.

Blood Examination	Sept. 30	Oct. 2
White blood cells	35,400	34,500
Polymorphonuclears	71	78
Lymphocytes	23	13
Mononuclears	6	9
Hemoglobin	52	

CASE 7.—Abscess of the lung (Fig. 8). Daniel D., aged 2 years. Admitted Oct. 29, discharged Dec. 31, 1913. Past history negative.

Present Illness.—Four months ago had pertussis followed one month later by a pneumonia complicated by a hydrothorax. This was treated by medicine and the condition was reported improved, although patient still suffered from occasional attacks of fever. Cough has been progressively worse. Child was taken to the outpatient department where a diagnosis of empyema was made.

Examination of the Lungs: Entire left chest, front and back, flat on percussion, absence of breath sounds.

Blood Examination:	Oct. 30	Nov. 21
White blood cells	17,000	38,000
Polymorphonuclears	72	76

Thoracotomy. No pus found in the pleural cavity, drainage tube left *in situ*. Three days later profuse discharge which continued for twenty-four days. Patient discharged on sixty-third day, wound closed. Normal breath sounds and resonance. Temperature for the first week ranged between 101 in the mornings to 103 to 104 in the afternoons, falling to 101 in the third week with slight exacerbations, and reaching normal in the fourth week.

CASE 8.—Abscess of the lung. Mary U., aged 6 years. Admitted July 10, discharged Aug. 18, 1913. Has had convulsions, measles, scarlet fever, pertussis and varicella.

Present Illness.—Three weeks ago tonsils and adenoids were removed. Following the operation there developed a high fever accompanied by a severe cough, which was often relieved by vomiting. The vomitus was brownish-yellow with a foul odor and was accompanied by a putrid expectoration. Mother thinks that the child has lost about six pounds in weight during the last ten days. For the past week patient has complained of severe pain in the left chest. At night there has been high fever.

Examination of the Lungs: In the left axilla, extending from the angle of the scapula to the base, there is dulness, absence of fremitus, voice and breathing.

Temperature on admission was 102, respirations 56, pulse 140. For the first three days the temperature ranged from 102 in the morning to 103 in the evening. It then fell to 101 with slight morning remissions for four days, after which it gradually became normal. The cough, which was very severe, occurred in paroxysms and was generally accompanied by foul-smelling vomitus or expectoration. After the cough often cavernous breathing could be heard over an area about the size of a dollar just below the spine of the scapula.

Blood examination:	
White blood cells	20,800
Polymorphonuclears	78
Mononuclears	5
Eosinophils	1
Hemoglobulin	74

Sputum Examination: Mucopurulent, few pus cells, few bacteria; no tubercle bacilli.

The course of the case is shown in Figures 8, 9 and 10. July 31, the date on which Figure 11 was taken, there was very slight cough and the patient had gained a pound in weight.

October 23: Patient has been very well since leaving the hospital and has gained six pounds in weight. Has very little cough or expectoration. Still has cavernous breathing at times just below the spine of the left scapula.

CASE 9 (Supplemenntary Case).—Pyopneumothorax (Figs. 12, 13 and 14). Through the courtesy of Dr. John Thacher the following case is reported from the adult medical service. M. P., female, aged 40 years. Admitted Nov. 21, 1912, discharged Jan. 22, 1913. Chief complaint, shortness of breath for the past seventeen days.

Present Illness.—For the past seventeen days patient has suffered from shortness of breath, pain in the chest accompanied by a severe cough. Two days ago the pain became more intense, especially on the left side, and the dyspnea increased.

Examination of the Lungs: Marked bulging and immobility of the entire left chest; right chest, 17⅛ inches; left chest, 17½ inches. No expansion on the left side. On this side fremitus is present over the root of the bronchi in front and behind, elsewhere absent. Hyperresonant tympanitic note is heard on percussion over the upper left chest in front and behind with dulness and flatness below. Breathing is distant and diminished with a slightly amphoric quality in the first space and in the supraclavicular triangle. Elsewhere it is completely absent. Occasional râle is heard. At the angle of the scapula voice has an exaggerated nasal quality; below this it is markedly diminished. Hippocratic succussion is present. Right chest shows dulness at the right apex, in front and behind, with bronchovesicular voice and breathing. Fremitus is increased and a few subcrepitant râles are heard at the right base in the axilla and posteriorly.

Heart: Entire heart is markedly displaced to the right; apex beat is neither seen nor felt. On percussion the area of cardiac dulness extends 2¾ inches to the right of the midsternal line in the third space, and 1½ inches to the left of the mid-sternal line in the fourth space. Maximum area of pulsation just below the ensiform cartilage, sounds are heard loudest over the ensiform. Action is regular, moderately rapid, sounds are distant but of fair quality. No accentuations or murmurs are heard. On admission temperature was 101.8, pulse 108, respirations 88. Patient very dyspneic, cyanotic, pulse of poor quality. Temperature ranged from 101.6 to 104 with morning remissions. Respirations 38 to 88. Pulse 108 to 130. On the second paracentesis, two days after admission, 450 c.c. of purulent exudate were withdrawn.

Blood Examination:	Nov. 22	Nov. 23
Red blood cells	5,000,000	
White blood cells	14,800	22,200
Polymorphonuclears	76	76
Lymphocytes	24	24

Red cells pale, anisocytosis and poikilocytosis.

Patient was referred to the surgical service where she was operated upon and a partial osteotomy of the eighth rib was performed. Patient was discharged cured Jan. 22, 1913.

CONCLUSIONS

The Roentgen ray is of distinct value in the diagnosis of diseases of the respiratory tract. In pneumonia a shadow may appear over the suspected area several days before the development of definite physical signs.

It is of value in determining the progress of lung involvement. Roentgenograms may be taken on successive days as long as there are signs of active advancement of the process.

The Roentgen ray offers material assistance in differential diagnosis, tuberculosis, abscess of the lung, lobar and bronchopneumonia.

Through the obliteration of the costal-phrenic angle, the Roentgen ray indicates the presence of an exudate.

I wish to express my thanks to Dr. Freeman for permission to report the cases from the Bliss ward; to Dr. Thacher and to Dr. Sumner for their courtesy in allowing me to use the medical histories, and to Dr. Leaming, the radiologist of the hospital, for the excellent photographs and for suggestions regarding their interpretation.

50 Central Park West.

CASES OF EPIDEMIC CEREBROSPINAL MENINGITIS TREATED WITH FLEXNER'S ANTIMENINGITIS SERUM

WILLIAM ALEXANDER SMITH, M.D.
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The following cases are placed on record because they show (1) the value of Flexner's antimeningitis serum in cases of epidemic cerebrospinal meningitis, even after the infection has lasted for many days or weeks; (2) the necessity for prolonged treatment in such cases, and on the other hand (3) the rapidity of cure when the serum is injected promptly after the manifestation of symptoms.

The first three cases reported were in children of one family.

CASE 1.—I. G., aged 8 months, was admitted to the hospital April 25, 1912. He was the youngest of four children. The onset of his present illness was April 16, 1912, with fever and irritability. When he was picked up he screamed with pain. After a continuation of these symptoms for five days, it was observed that he did not use his right arm. He remained fretful, his paralysis and fever continued, and for these reasons he was brought to the hospital, but chiefly because his sister, who was taken ill after him with a much more severe form of the disease, had already been admitted and had begun to show improvement from treatment. On examination in the ward (April 25, 1912), there was extreme hyperesthesia and he was exceedingly irritable. There was a paresis of the right arm, a *tache cérébrale* and exaggerated reflexes at the knee—*Kernig's sign was absent, the neck was not rigid and the anterior fontanel not bulging*—the temperature was 102.5 F. The leukocytes were 34,000.

Lumbar puncture No. 1 was done shortly after admission. Thirty c.c. of turbid fluid were removed and 20 c.c. of Flexner's serum were injected. The fluid was turbid and under increased pressure. The cells were mainly polymorphonuclear leukocytes and contained Gram-negative diplococci. The meningococcus was obtained from the fluid in culture. The Noguchi globulin reaction was positive. No unfavorable symptoms followed the injection of the serum.

The next morning (April 26, 1912) the temperature was 99 F., but as there was no other appreciable improvement in the child's condition, *lumbar puncture No. 2* was done; 20 c.c. of fluid were removed and 20 c.c. of serum were injected. The fluid was cloudy. There were many pus cells but no organisms were found. Culture showed no growth. After this injection the temperature rose to 105 F., but in twelve hours had fallen to 99 F.

The following day (April 27, 1912), as there was still no improvement in the general condition, *lumbar puncture No. 3* was done; 60 c.c. of fluid were removed and 15 c.c. of serum injected. The fluid was clear. There were very few pus cells and no organisms were found. There was a temperature reaction to 104 F. following this injection. The child showed improvement in the next twenty-four hours by being less irritable and less hyperesthetic. The right arm was still helpless, but the reflexes at the knee were not exaggerated. *Lumbar puncture No. 4* was done on this day (April 28, 1912); 15 c.c. of fluid were removed and 15 c.c. of serum injected. The fluid was clear and contained no pus cells or organisms. The temperature rose to 103 F. after this injection. The leukocytes were 15,000.

Lumbar punctures Nos. 5, 6 and 7 were done on the next three days and 20, 30 and 50 c.c. of clear fluid, respectively, withdrawn, containing no pus cells and no organisms. The punctures were performed in order to see if organisms were present, in which case serum was to be again injected, but also because each time, with a considerable accumulation of fluid, there was restlessness and irritability. These symptoms were at once relieved by withdrawing the excess of fluid. The child's improvement was marked, but as the temperature rose to 102 F. on May 2, 1912, *lumbar puncture No. 8* was done and 20 c.c. of fluid were obtained. The fluid was cloudy and contained many pus cells. For this reason 20 c.c. of serum were injected, but no organisms were found and the

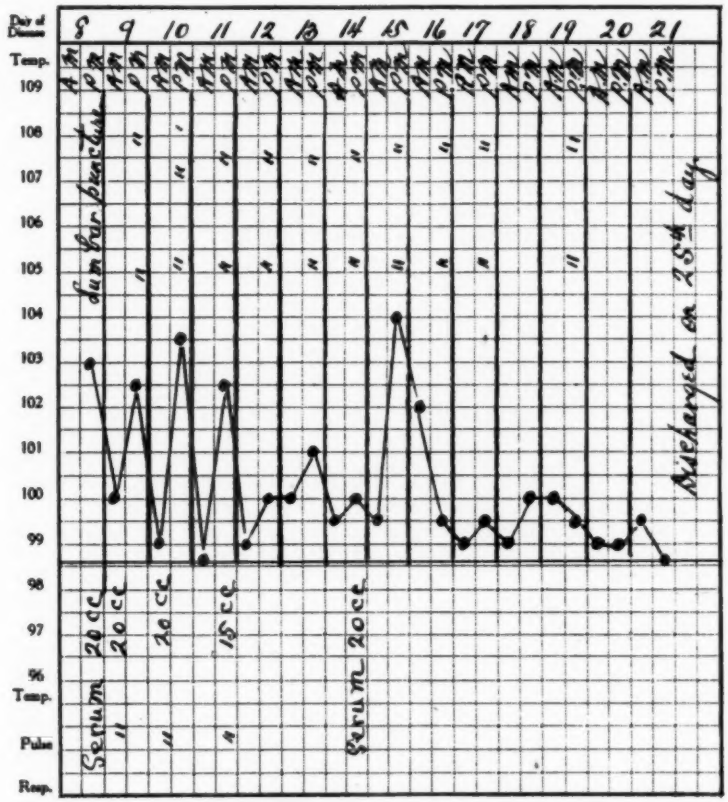

Chart 1.—Temperature curve in Case 1.

cultures were negative. The leukocytes were 24,000. The temperature rose to 104 F. after the injection. *Lumbar punctures Nos. 9 and 10* made on the following two days showed slightly cloudy fluid with few pus cells, but no organisms. The Noguchi globulin reaction was still positive.

Lumbar puncture No. 11 was made two days later (May 6, 1912); 10 c.c. of fluid were obtained. This was clear. There were no pus cells and the Noguchi reaction was negative. The leukocytes were 12,000.

From this time the child improved rapidly. The temperature remained normal and the power in the right arm returned. He was discharged May 11, 1912, entirely well.

In this case the hyperesthesia, marked irritability of the child and the paresis of the right arm, were strongly suggestive of meningitis even in the absence of rigidity of the neck, Kernig's sign and bulging of the fontanel. It is interesting to observe that four days after the last injection of serum the spinal fluid became cloudy and that there were many pus cells but no organisms.

The reactions due to the serum were definite and sharp but were of short duration and entirely without sequelae.

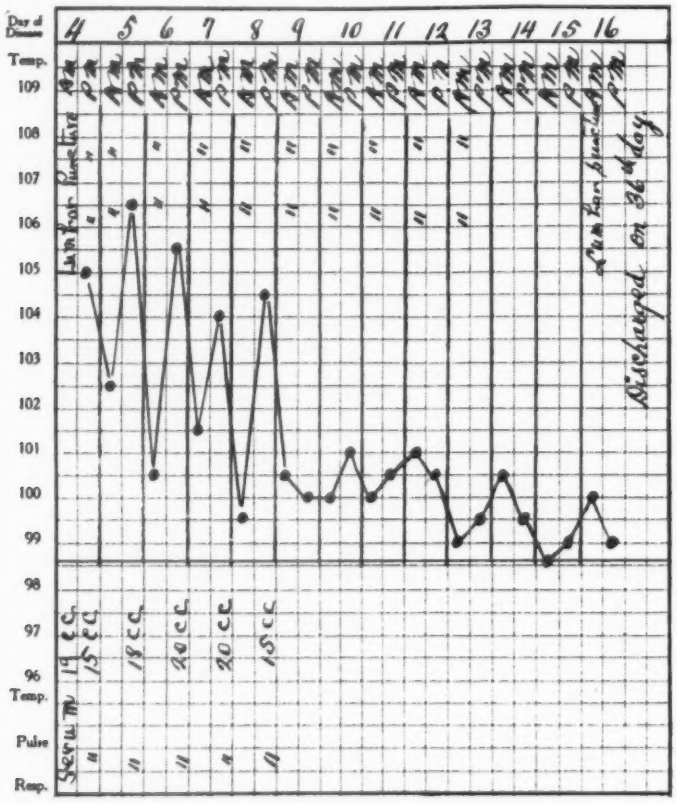

Chart 2.—Temperature curve in Case 2.

CASE 2.—F. G., aged 2½ years, was admitted to the hospital April 24, 1912. The onset of the present illness began April 20, 1912, four days after that of her brother (Case 1). She was taken acutely ill with fever, vomiting, headache in the left frontal region and convulsions. The next day (April 21, 1912) there was in addition a petechial eruption about the face and chest. Her condition remained the same during the next two days and on admission to the ward, April 24, 1912, she was stuporous and having one convulsion after another. There was slight rigidity of the neck muscles, internal strabismus of the right eye, a coarse tremor of the extremities, exaggerated reflexes, a *tache cérébrale*, a bilateral Babinski and a positive Macewen's sign. Kernig's sign was not present. The temperature was 105 F.; the leukocytes 28,000.

Lumbar puncture No. 1 was done at once; 20 c.c. of thick purulent fluid were withdrawn and 15 c.c. of Flexner's serum injected. The fluid showed many pus cells and Gram-negative intracellular diplococci. The meningococcus was obtained by culture. The Noguchi globulin reaction was positive. There was a severe reaction after the injection of the serum, as shown by the rise of temperature and an increase of the nervous symptoms. This lasted about one hour.

The child's general condition improved after the first injection and the next day, April 25, 1912, she could be aroused, the temperature was 101 F. and there were no convulsions.

Lumbar puncture No. 2 was done; 35 c.c. of fluid were withdrawn and 20 c.c. of serum injected. The fluid was turbid and contained many pus cells, but few organisms. The cultures were positive. After this injection of serum there was a severe reaction. The temperature rose to 107 F., the respiration was irregular and rapid and there was a coarse general tremor. No convulsions.

On the following day (April 26, 1912) the temperature was 100 F., and although she lay for the most part in a stupor she recognized her parents when aroused. The reflexes were not exaggerated and there were no convulsions. *Lumbar puncture No. 3* was done; 35 c.c. of fluid were removed and 20 c.c. of serum injected. The fluid was cloudy. There were many pus cells but only a few organisms. The organisms were extracellular. The cultures showed no growth. The Noguchi reaction was positive. There was the same severe reaction lasting for about one hour after this injection.

April 27, 1912, although the child was in every respect improved, *Lumbar puncture No. 4* was done; 35 c.c. of fluid were obtained and 20 c.c. of serum injected. The fluid was clear, containing only a few pus cells and no organisms. The Noguchi reaction was positive. The reaction after this injection was not severe except for the high temperature.

Lumbar puncture No. 5 was done April 28, 1912; 20 c.c. of clear fluid containing no organisms were obtained; 15 c.c. of serum were injected. The Noguchi reaction was negative. *Lumbar puncture* was done the five days following. Twenty to 70 c.c. of clear fluid containing no pus cells or organisms were obtained. After an interval of two days 80 c.c. of clear fluid were obtained by *lumbar puncture No. 11.* This fluid showed no pus cells or organisms. The Noguchi reaction was negative. The indications for the repeated withdrawal of fluid were the same as with the first child. The child improved rapidly after the fifth injection of serum. The temperature varied between 99 F. and 101 F., gradually becoming normal. The leukocytes fell to 12,000 and except for a serum rash ten days after the first injection of serum, the convalescence was uneventful. During convalescence lumbar punctures, four in all, were done at intervals. Each time from 10 to 30 c.c. of clear fluid were obtained.

The case was a severe one. Four days after the onset the child was in a stupor, the temperature was very high and she was having frequent convulsions.

CASE 3.—M. G., aged 6 years, was taken ill April 26, 1912, at noon, ten days after her brother (Case 1). The onset was acute, with vomiting, frontal headache and fever. She was admitted to the hospital four hours later (4 p. m., April 26, 1912) after having had several convulsions. On admission the temperature was 103 F., there was drowsiness and she complained, when aroused, of intense headache. On examination there was a *tache cérébrale,* slightly exaggerated reflexes and dilated pupils. Macewen's sign was present. There was no rigidity of the neck muscles, nor was Kernig's sign present. The leukocytes were 30,000.

Lumbar puncture No. 1 was done at once; 25 c.c. of fluid were withdrawn and 20 c.c. of Flexner's serum injected. The fluid was clear but contained, after centrifugation, a few pus cells and Gram-negative intracellular diplococci. The cultures showed meningococci. The Noguchi globulin reaction was positive.

The following day (April 27, 1912) there was a fine petechial eruption on the upper part of the chest and neck. The patella tendon reflexes were markedly exaggerated and Kernig's sign was present. In other respects, however, there had been a marked improvement. The temperature was lower (100 F.) and the child was much brighter and complained no longer of headache. Lumbar puncture was not done. From this day improvement was rapid. The temperature remained normal and the number of leukocytes fell to 12,000 (May 7, 1912). Convalescence was uneventful except for the appearance of an urticarial eruption on the seventh day and the child was discharged May 7, 1912, eleven days after the onset of the disease.

These three cases show almost with the nicety of a carefully planned experiment the value of the serum treatment and the rapidity of its effect in direct proportion to the time of its employment. Children

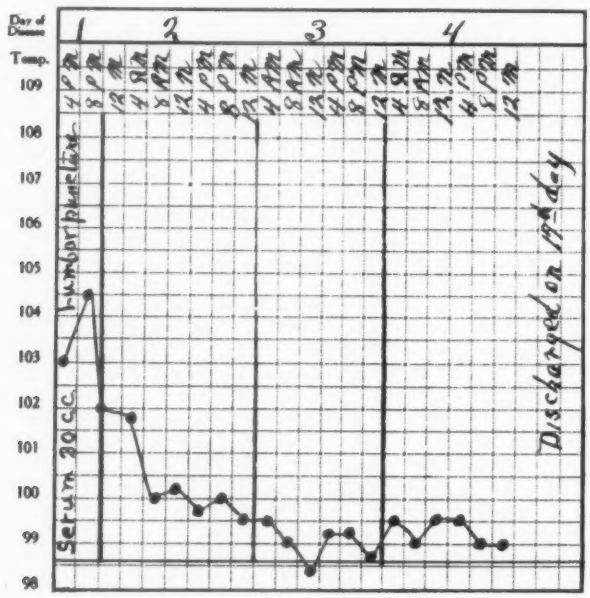

Chart 3.—Temperature curve in Case 3.

1 and 2 had been ill nine and four days, respectively, before the institution of treatment. Both were severely ill, the second child particularly so. In both the disease yielded to treatment, but only after many days and repeated injections of the serum. In marked contrast is the case of Child 3, a member of the same family. Her disease was marked by a sudden and severe onset, yet it yielded promptly and permanently to only one injection of the serum. I know of no similar recorded case in which treatment was begun so soon (four hours) after the beginning of symptoms. The temperature charts show graphically this distinction. Of particular interest was the fact that the duration of the disease was so short that the cerebrospinal fluid had not yet

become turbid. Such a fluid though unusual is occasionally encountered. The presence of meningococci, together with the perfectly characteristic nervous symptoms and the petechial eruption, leave no doubt of the diagnosis. Unless other children in the same family had previously suffered from similar symptoms, it is likely that hours or days would have elapsed before serum treatment would have been possible.

The two following cases are examples of prolonged meningococcus meningitis showing no tendency to improve, but yielding at once to the serum:

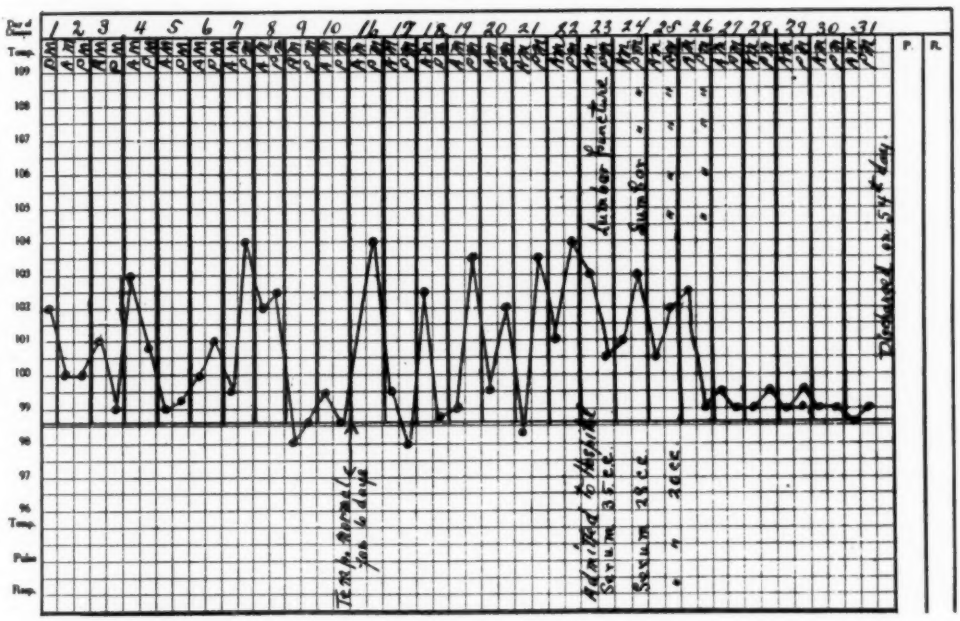
1 2 3 4 5 6 7 8 9 10 16 17 18 19 20 21 22 23 24 25 26 27 28 29 30 31
Lumbar Puncture
Serum
Discharged on 54th day.
Temp. Normal for 6 days
Admitted 6th day
Serum 35 c.c.
Serum 28 c.c.
" " 20 c.c.

Chart 4.—Temperature curve in Case 4.

CASE 4.—L. A., aged 4 years, was admitted to the hospital May 23, 1912. The present illness began April 30, 1912, with fever, convulsions and headache. The temperature varied intermittently from 97 to 104 F. and the convulsions continued at irregular intervals until May 9, 1912. Then the temperature remained normal and he was up and about the house until May 15, 1912, a period of six days. On the fifteenth he had a convulsion and the temperature rose to 104 F. The intermittent temperature and convulsions continued and he was admitted to the hospital May 23, 1912, twenty-three days after the onset of the disease.

On examination in the ward he was found to be greatly prostrated, emaciated, irritable and complained of a severe headache. There was marked hyperesthesia, rigidity of the neck, exaggerated reflexes, ankle clonus and a positive Kernig and Macewen sign. At night he had a shrill meningeal cry; the temperature was 103 F. The leukocytes were 24,000. *Lumbar puncture No. 1* was done; 25 c.c. of fluid were withdrawn. The fluid was cloudy and

contained pus cells and many Gram-negative intracellular diplococci. Cultures showed meningococci. The Noguchi globulin reaction was positive; 25 c.c. of Flexner's serum were injected six hours later. During the injection of the serum the child complained of severe pain in the legs and the temperature which was 97 F. afterwards rose to 103 F.

The next day, May 24, 1912, he showed improvement by being less hyperesthetic and less irritable. The temperature remained high. *Lumbar Puncture No. 2* was done; 50 c.c. of fluid were removed and 40 c.c. of serum were injected. The fluid was slightly cloudy and contained few pus cells, but no organisms. The Noguchi reaction was positive.

Lumbar puncture No. 3 was done May 25, 1912; 25 c.c. of fluid were withdrawn and 20 c.c. of serum injected. There was a reaction in the temperature to 103 F. The fluid removed at this time was collected in four test tubes. It flowed freely from the needle, and though clear, had a peculiar yellow hue. It was taken immediately, after the serum had been injected, to the laboratory, and there was found to have coagulated to the consistency of solid agar. There were no pus cells nor organisms. The cultures showed no growth.

Lumbar puncture No. 4 was done the next day, May 26, 1912. The fluid appeared in drops at the end of the needle and coagulated at once, and when a drop was wiped off another would appear and rapidly become coagulated. At this time the child showed marked improvement, the temperature remained normal, and except for a great weakness which continued to the time of his discharge (June 23, 1912) he made an uneventful recovery.

CASE 5.—E. T., aged 6 years, was admitted to the hospital Nov. 27, 1912. The present illness began three weeks previously (Nov. 7, 1912) when she complained of headache and feeling tired. She was in this state for about one week or until Nov. 15, 1912, when she had three general convulsions, each lasting about five minutes. After that she remained in bed with headache and an afternoon rise of temperature to 104 F. One week after this (Nov. 20, 1912) her neck became stiff. Since then she had had headache, afternoon fever and stiffness of the neck. She had not vomited. On admission to the ward she was conscious and complained of headache. There was extreme emaciation, the head was drawn back and the back arched. Macewen's sign was not present. The eye grounds were normal. The abdomen was boat-shaped. There was a bilateral Kernig, exaggerated reflexes and ankle clonus. The temperature was 100 F., the leukocytes 30,400.

Lumbar puncture No. 1 was done; 20 c.c. of fluid were removed and 20 c.c. of Flexner's serum injected. The serum was turbid and under increased pressure. There were many pus cells and Gram-negative intracellular diplococci and the Noguchi reaction was positive. The meningococcus was obtained by culture. After the injection the temperature rose to 104 F. The next day (Nov. 28, 1912) there was no improvement and *lumbar puncture No. 2* was done; 15 c.c. of fluid were removed and 20 c.c. of serum injected. The fluid was turbid and there were a few pus cells with a few Gram-negative intracellular diplococci. Cultures showed the meningococcus. The temperature fluctuated for the next few days from 99 F. to 104 F., but there was no improvement in the child's condition. *Lumbar punctures Nos. 3, 4 and 5* were done and 20, 40 and 15 c.c., respectively, of serum were injected. There was no growth on cultures and the fluid was clear after the second injection of the serum. The Noguchi reaction remained positive. After the fifth injection, Dec. 1, 1912, the temperature remained normal. The child's general condition was improved and she could move her neck freely from side to side. The retraction continued but was less marked. Dec. 5, 1912, the temperature rose to 103 F., and though she seemed in as good condition as before, *lumbar puncture No. 6* was done; 30 c.c. of fluid were removed and 30 c.c. of serum injected. The fluid was clear; there were no pus cells or organisms. The cultures were

negative and the Noguchi reaction was negative. The rigidity of the neck became less and less, disappearing about Dec. 11, 1912. Convalescence was slow and she was discharged Dec. 26, 1912.

DISCUSSION

These five cases varying in duration from four hours to twenty-three days were all favorably influenced by the antimeningitis serum. All the patients recovered without sequelae. A study of these prolonged

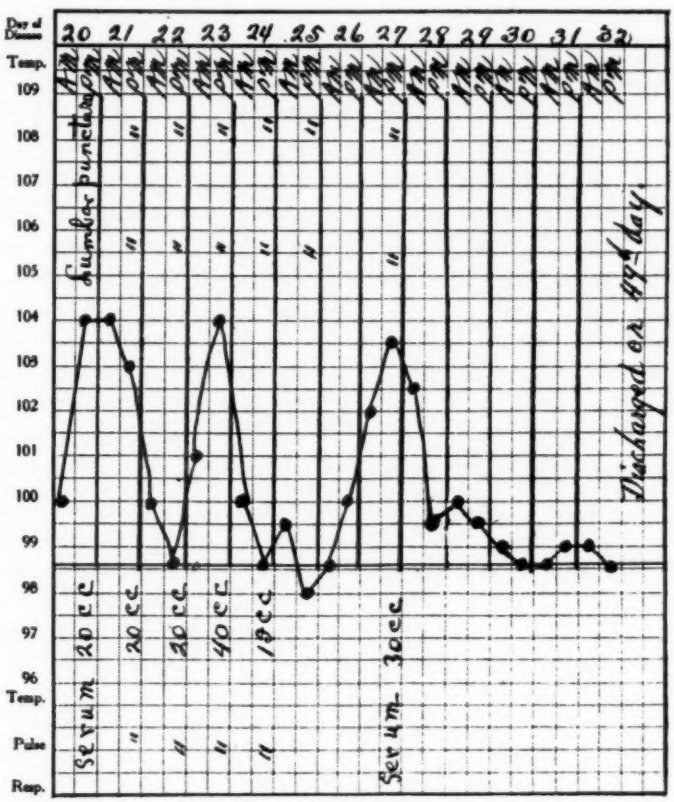

Chart 5.—Temperature curve in Case 5.

cases shows the necessity of repeated injections of the serum and of continuing the withdrawal of cerebrospinal fluid at frequent intervals well after the fluid is clear and no organisms are to be found microscopically or by culture. The nervous symptoms and their prompt amelioration after lumbar puncture make this clear. That such large quantities as 20 to 80 c.c. of fluid can be withdrawn shows that temporarily the injury to the meninges prevents the absorption of the fluid which is undoubtedly formed in excess owing to inflammatory irritation of the choroid plexus.

Occasionally the fluid may exhibit unexpected and peculiar characteristics, as in Case 4, in which spontaneous coagulation took place, and in Case 1, in which in convalescence a cloudy fluid due to polymorphonuclear leukocytes appeared without any cocci or other organisms being present and following several findings of perfectly clear fluid.

The reactions due to the repeated injections of foreign serum were marked. There was frequently high fever and restlessness, but these were of short duration.

These cases while few in number serve nevertheless to emphasize the fact that in any case of meningococcus meningitis, no matter of what duration, provided viable meningococci are present, Flexner's antimeningitis serum should be used repeatedly, and that even in apparently unfavorable cases, complete recovery may occur.

The first four cases were observed in the St. Louis Children's Hospital in the service of Dr. Howland, and I am further indebted to him for the opportunity of reporting the fifth case.

122 West Lockwood Avenue.

A STUDY OF 105 CASES OF TUBERCULOUS MENINGITIS*

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SAN FRANCISCO

Most of the leading text-books on pediatrics give very good composite pictures of tuberculous meningitis, but, as in many of the other diseases, one rarely sees the whole picture in any one case. Nor has he any idea of the relative frequency of the various signs and symptoms of the disease. It was the intention, in the analysis of these cases, to present everything that appeared in the histories, thereby giving an idea of the disease as it appears on the records of the Children's Hospital in Boston. The analysis comprises all the cases which were admitted to the hospital since January, 1910, which takes it back before a few of the later signs of meningitis were known. Most of the signs are more or less present at some stage of the disease, and it is interesting to note at just which stage any particular sign appears and disappears. Many of the records are complete, and while in not a few of the cases neither the organism nor the necropsy was obtained, yet the diagnoses were unquestionable from the clinical picture and were made by competent men.

Sex.—Of the 105 patients, 53 were females and 52 were males, the youngest being 5 months old and the oldest 11 years. Over 30 per cent. were between the ages of 2 and 3 years, while over 56 per cent. were between the ages of 2 and 5 years and over 80 per cent. between the ages of 18 months and 5 years (see Chart 1). This is interesting from the fact that most of the other observers have recorded the ages up to 3 years of age, while Chart 1 shows the relative frequency up to 12 years of age, which is the age limit in the Children's Hospital. No case occurred in patients under 5 months or over 11 years in the series. Holt's[1] largest number came between the ages of 9 and 12 months (20 per cent.).

Seasonal Prevalence.—Holt, in 1911, showed that the seasonal prevalence for tuberculous meningitis in New York, was in the month of April, 16 per cent. of his 218 cases occurring in that month. Recording all the cases which were admitted into the Children's Hospital for the last ten years, a total of 273 cases, Boston's highest number occurred

* Received for publication Feb. 25, 1915.

* This work was done at the Children's Hospital, Boston, during the service of the late Dr. T. M. Rotch.

1. Holt, L. Emmett: Observations on Three Hundred Cases of Acute Meningitis in Infants and Young Children, AM. JOUR. DIS. CHILD., January, 1911, p. 26.

during the months of June, 12.3 per cent., July, 11.3 per cent., and January, 11 per cent., while April produced only 9.5 per cent. October and November produced the lowest number, 5.8 per cent. (see Chart 2). Computing the seasonal prevalence for the last four years, as seen in Chart 3, the largest number occurred during the month of January, 15.2 per cent., followed by the month of April with 10.5 per cent., and July, 9.5 per cent., while the lowest percentage occurred during the months of September, October and November, 5.7 per cent. Up to 1909 there had been a gradual increase in the number of cases recorded (Chart 4). Since that time there has been a gradual decline, undoubtedly due to the improved methods of sanitation, the reporting of all tuberculous cases, the influence of social service, in short, to the awak-

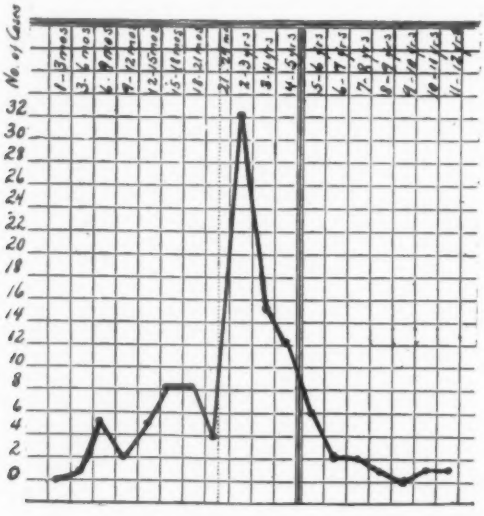

Chart 1.—Showing ages when the disease was contracted.

ened interest in tuberculosis. Tabulating the seasonal prevalence for babies under 16 months (Chart 5), there were more cases during the months of January and March than in any other month.

Previous Diseases.—Measles was mentioned in the past history of twenty-seven cases, or 26 per cent., and whooping-cough in 22 per cent. Other diseases mentioned in the histories were as follows: pneumonia, 11 per cent.; chicken-pox, 8 per cent.; bronchitis, 6 per cent.; diphtheria, 6 per cent.; mumps, 4 per cent.; otitis media, 4 per cent.; colds, 2 per cent.; Pott's disease, 2 per cent.; tuberculous osteomyelitis 1 per cent.; indigestion, 1 per cent.; abscesses, 1 per cent.; tuberculous lymph-nodes, 1 per cent.; coryza, 1 per cent.; malaria, 1 per cent. Some of the patients had had only one disease, while others had had four or five. Thirty-eight per cent. had had no previous mentioned diseases.

The most frequent combinations of previous diseases were: measles and whooping-cough in three cases, measles and pneumonia in three cases, and measles and chicken-pox in two cases. Other combinations were as follows: measles, chicken-pox, whooping-cough and suppurative adenitis; whooping-cough and diphtheria; measles, bronchitis and otitis media; whooping-cough, diphtheria and chicken-pox; whooping-

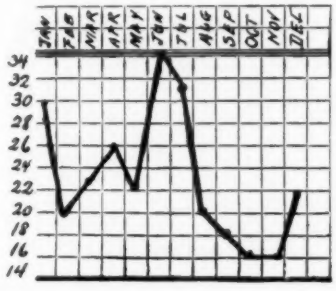

Chart 2.—Seasonal prevalence of 273 cases of tuberculous meningitis.

cough and adenitis; whooping-cough and bronchitis; whooping-cough, measles and mumps; whooping-cough, measles and pneumonia; whooping-cough, measles, mumps, bronchopneumonia, laryngeal diphtheria and otitis media; whooping-cough and pneumonia; measles, mumps and chicken-pox; measles, pneumonia and otitis media.

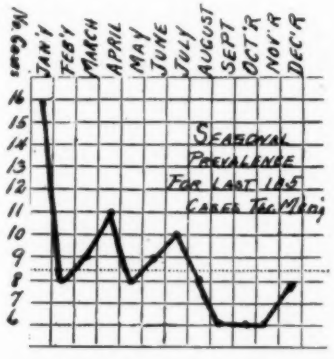

Chart 3.—Seasonal prevalence for last 105 cases tuberculous meningitis.

Duration.—Holt found that the average duration of the disease of children up to 3 years of age, from the time the child began to show signs of apathy, vomiting and slight temperature, was two and a half weeks. Rankin[2] gives twenty-three days as the duration for his series. The average duration of the disease found in this series of all cases of

2. Rankin, G.: Meningitis in Children, Brit. Med. Jour., London, 1910, i, 1045.

children up to 12 years of age, from the beginning of symptoms, was seventeen days, which is exactly the same as Holt reported for children under 3 years in New York. The shortest duration was five days, while the longest duration was forty-three days. Nearly 50 per cent. of all the patients were sick from ten to eighteen days (Chart 6). The duration seems to vary somewhat in the different ages. Chart 7 shows

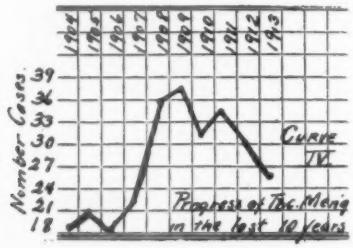

Chart 4.—Progress of tuberculous meningitis in the last ten years.

that the average duration of the disease is shorter in a child past 4 years of age, being greatest between the ages of 3 and 4 years (19.5 days) and shortest between the ages of 4 and 12 years (15 days).

Duration in the Hospital.—The average duration in the hospital was 5.8 days, the longest being 21 days and the shortest 12 hours. Only 14 per cent. of the patients lived 10 days or over in the hospital, and

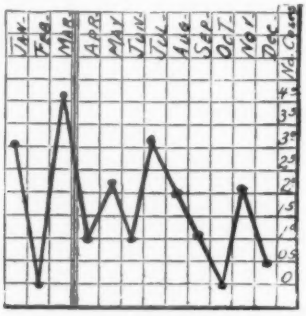

Chart 5.—Mortality under 16 months.

nearly 60 per cent. lived 5 days or less. Chart 9 shows that most of the patients lived 3 days. The duration of the disease is longer now than formerly, probably owing in part to the earlier diagnoses and repeated lumbar punctures. That the latter is true may be seen from Chart 8, the average duration in the hospital varying almost directly with the number of lumbar punctures, ranging from 10 to 14 days with five lumbar punctures, to 4.4 to 9.7 days with four lumbar punctures or

under. This fact lends support to Dunn's suggestion[3] of early and repeated lumbar punctures as part of the treatment of tuberculous meningitis. In one of the very recent cases in the hospital the patient

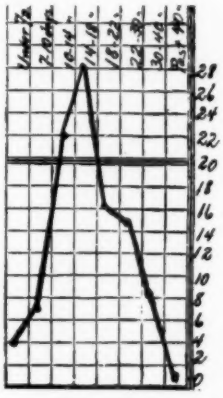

Chart 6.—Duration of disease.

had seven lumbar punctures and lived 15 days, having been sick 22 days outside the hospital. In the last ten cases in the hospital the patients had on an average 4.4 lumbar punctures each, while the average duration was 9 days. In thirty consecutives cases previous to these the

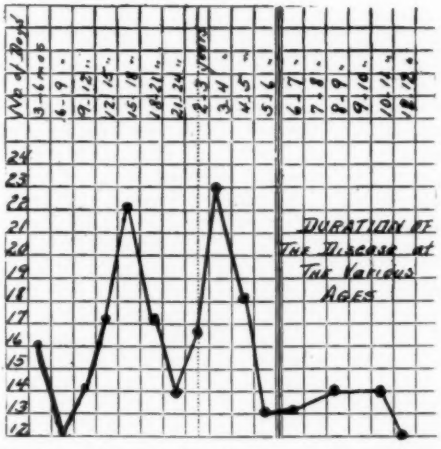

Chart 7.—Duration of the disease at the various ages.

patients had on an average 1.9 lumbar punctures, while the average duration was only 5.4 days.

Lumbar Punctures.—The largest number of lumbar punctures made in one case was seven, and the smallest one, puncture being often

3. Dunn, Charles Hunter: Cerebrospinal Meningitis, Its Etiology, Diagnosis, Prognosis and Treatment, Am. Jour. Dis. Child., February, 1911, p. 95.

made simply for diagnosis. Lumbar puncture was done once in each of 58 cases, twice in each of 26 cases, three times in each of 6 cases, four times in each of 5 cases, five times in each of 8 cases, and seven times in each of 2 cases. Most of the patients receiving more than four lumbar punctures were the recent ones. Many of the patients seemed to brighten up after the withdrawal of spinal fluid, and in not

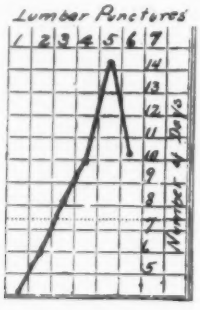

Chart 8.—Relation of number of lumbar punctures to duration.

a few cases there followed a remission of pulse and temperature. Convulsions in tuberculous meningitis at the present time in the hospital are almost unknown since spinal puncture has become part of the treatment of the disease. In no case was there any record of any harm having been done owing to the withdrawal of too much spinal fluid.

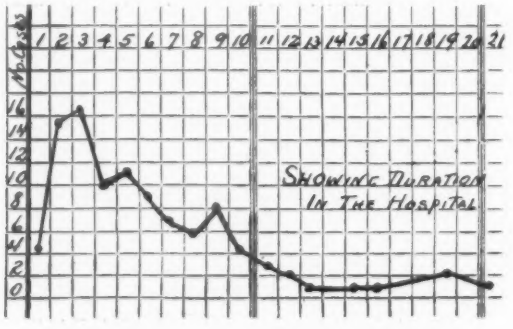

Chart 9.—Duration in the hospital.

The spinal canal was allowed to drain until the fluid ran at the rate of about ten or twelve drops per minute. In view of the two cases reported by Stiles[4] before the International Congress on Tuberculosis at Washington in 1908 as undoubted cures, also the rapidity with which the spinal fluid recurs under this pathologic condition, the appar-

4. Stiles, C. W.: Proc. Sixth Internat. Cong. on Tuberculosis, Washington, 1908.

ent brightening of the patient after each lumbar puncture, the elimination of a certain amount of toxin, and also the fact that the spinal fluid in repeated punctures is generally under but very slight pressure, it would seem that there is but very little danger, if any, in removing too much spinal fluid from the canal. As a general rule, there is rarely any difficulty in holding a child as there is in doing a spinal puncture in the epidemic form, nor is there sufficient pain to justify the discomfort of anesthesia. Usually at the time when the child enters the hospital there is scarcely any rigidity of the spine, and the degree of apathy is so marked that it is a perfectly simple procedure to draw off

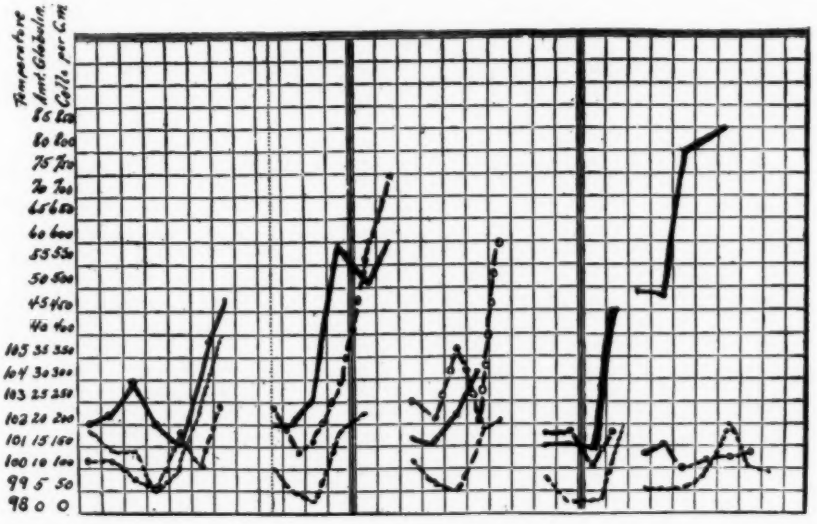

Chart 10.—Relation between amount of globulin, temperature and cells per cubic millimeter. Solid line denotes amount of globulin, dotted line, temperature and line composed of dashes and circles, cells per cubic millimeter.

from 20 to 30 c.c. of fluid as the case permits. Lumbar puncture is done in all cases once every forty-eight hours.

Organisms in the Spinal Fluid.—Several articles on the examination of spinal fluid in tuberculous meningitis have been published, in which it is reported that the organism has been found in practically every case. As a matter of fact, the finding of tubercle bacilli in spinal fluid resolves itself into a matter of time. With careful technic, it has been possible to demonstrate the organisms nearly every time in the first or second puncture, but the third or fourth puncture almost invariably shows them. The system in vogue then at the hospital was to centrifugalize the fluid with water-power for from six to twelve hours. All but the last three or four drops of fluid plus the clot was drawn off with a long sterile dropper and thrown away. The clot and the remaining

fluid were then put on a cover-slip and teased out with fine sterile needles. This cover-slip preparation was then allowed to dry in the air, fixed, and then stained with carbol-fuchsin, decolorized with 20 per cent. sulphuric acid and counter-stained with Loeffler's methylene blue. Tubercle bacilli were found in 21.5 per cent. of this series of cases, and were found just as readily in infants as they were in older children. Seventeen per cent. of the cases were verified by necropsy, no organisms having been found, which was evidently the reason for necropsy. This makes a total of 38.5 per cent. which were found to be positively tuberculous. Hemenway[5] notes that the last fluid withdrawn after the child is raised to the sitting position is the best to examine for organisms. This is probably due to the fact that the last part withdrawn seems to contain more fibrin than the first part. The relation of the first part of the spinal fluid to the last part will be taken up later.

Pressure and Clearness of Spinal Fluid.—The spinal fluid was under pressure in 80 per cent. of the cases recorded, varying all the way from very slight to very great pressure. In 11 per cent. of the cases the pressure was normal. In 9 per cent. it was not recorded. In most cases it appeared late in the disease. Where the pressure was normal the amount usually did not exceed 20 c.c. of fluid. The amount of pressure, as roughly gauged, bore no relation to the age or to the abolition or retention of reflexes. Broadly speaking, however, where there was no pressure, there was no or scarcely any disturbance of the reflexes. There was no relation between the amount of pressure and the presence of convulsions, bulging fontanelle or retraction of the head. Most of the fluid obtained was clear, few of the cases recording "very slight turbidity" or "contained small bits of fibrin." A few were recorded as blood-tinged, and one was contaminated by secondary infection. In no case was the fluid recorded as actually turbid. The average amount of fluid obtained was 25 c.c., and ranged from 5 c.c. to 75 c.c .

Cell Count.—The average cell count was 198 cells to the cubic millimeter, the lowest counts being 24, 26, 30, 35, 38, 48, and 50 cells, while the highest count was 960 cells. There was no relation between the cell count and the age, sex or amount of spinal fluid. Its relation to the globulin content of the spinal fluid and the temperature and the stage of the disease will be discussed under the head of globulin. The cell count seemed to bear some slight relation to the white blood count,

5. Hemenway, Josephine: The Constant Presence of Tubercle Bacilli in Cerebrospinal Fluid of Tuberculous Meningitis, AMER. JOUR. DIS. CHILD., January, 1911, p. 37.

for the average white blood count for all cases with cell counts over 350 cells, was 22,700, while the average white count for all cases with cell counts under 100 cells, was 17,000. The prevailing type of cell in the spinal fluid was the small mononuclear, ranging from 90 to 100 per cent. in 67 per cent. of the cases, and from 80 to 90 per cent. in 20 per cent. of the cases. In 4 per cent. of the cases the mononuclears ranged between 70 and 80 per cent., in 3 per cent. the mononuclears ranged between 60 and 70 per cent., and in 3 per cent. the mononuclears ranged between 40 and 60 per cent. There were only three cases in which the polymorphonuclear leukocyte was the prevailing type of cell, one recording 85 per cent. polymorphonuclears, and in two others "mostly all the cells were polymorphs."

Fibrin Clot.—A definite fibrin clot was present in 70 per cent. of the cases, absent in 8 per cent., and not mentioned in 22 per cent. The time of appearance of the clot varied from one to forty-eight hours. The clot seemed to form earlier in the last part of the spinal fluid than it did in the first part of the same puncture.

The Noguchi or Butyric Acid Test for Globulin.—A positive test for globulin was obtained in 50 per cent. of the cases; in 7 per cent. it was negative, and not recorded in 43 per cent. There is no doubt but that there is a positive globulin test in every case of tuberculous meningitis. In the 7 per cent. of negative results, there must have been an error in technic, or enough time was not given for the precipitation of globulin, or else there was a lack of familiarity with the normal globulin content of the spinal fluid. Normal spinal fluid, when treated with 10 per cent. butyric acid in normal saline, one to five parts, heated to boiling, will, on the addition of one part of normal sodium hydroxid, give a slight turbidity, which rarely precipitates on standing twenty-four hours. Tuberculous meningitic fluid will, in practically every case, give a precipitation almost immediately, especially if boiled after the normal sodium hydroxid is added. Some of the cases show only a slight increase over the normal, and if one is not in the habit of measuring the globulin, he may erroneously call it a negative test. The amount of globulin seems to bear a certain relation to the cell count and temperature, and follows them pretty closely (see Curves A, B, C, D, and E of Chart X). Comparing five cases of tuberculous meningitis, in which the globulin content was measured by volume each time a spinal puncture was done (every forty-eight hours in each case) and an average cell count made of the fluid, one notes a close relationship. It was not until the fourth case was studied that a difference was noted between the first part of the spinal fluid and the last part withdrawn at the same puncture. Undoubtedly, if averages had been taken in all the

five cases, the curve of the globulin content would follow the curve of the cell count precisely. In all the cases the cell count was the highest at the last puncture, likewise the amount of globulin was greatest. In nearly every case both dropped at just about the time when the temperature made its final drop. There seemed to be no constant relation between the number of cells in the spinal fluid and the amount of globulin in different cases; that is, one case may have a count of 150 cells per cubic millimeter and have 10 per cent. of precipitated globulin by volume, while another case may have fewer cells per cubic millimeter and have almost 50 per cent. of precipitated globulin by volume, both cases being examined the same number of days before the end. The epidemic form of meningitis may have a cell count of 5,000 cells per cubic millimeter with only 20 to 30 per cent. of precipitated globulin by volume. The amount of globulin does not seem to bear any relation to the age. As regards the percentage of globulin, it seemed fairly constant in every case but the last, that of a child of 8 months, which showed almost three times as much as the other cases, in each of which the patient was older. The curves A and C of Chart X are almost identical, the children being of nearly the same age. It is impossible to make any generalizations from only five cases, and one can merely hint at the conclusions which they seem to present. Four out of the five cases had white blood counts below 16,400, and the one with the highest amount of globulin had a white count of 26,000.

The first part of the spinal fluid withdrawn from the spinal canal in cases of tuberculous meningitis differs materially from the last part taken at the same puncture. There is a marked difference in the number of cells, as will be seen in the following cell counts. These figures are the average of five different counts.

First 10 c.c. Drawn	Last 10 c.c. Drawn
183 cells per c.mm.	100 cells per c.mm.
190 cells per c.mm.	120 cells per c.mm.
133 cells per c.mm.	76 cells per c.mm.
124 cells per c.mm.	88 cells per c.mm.
206 cells per c.mm.	160 cells per c.mm.

The excess of cells in the first part cannot be due to gravity, the child lying usually with its head higher than its pelvis, because other constituents of the spinal fluid, which are in solution, differ accordingly. In some instances there was nearly twice as much globulin in the first part as there was in the last. In the patient 8 months old there was 0.9 c.c. of precipitated globulin in 2 c.c. of the first part of the spinal fluid withdrawn at one of the last punctures, and 0.7 c.c. of precipitated globulin in 2 c.c. of the last part withdrawn at the same puncture. One

of the earlier punctures in the same case showed 0.66 c.c. precipitated globulin in the first part and 0.36 c.c. in the last part. This difference was constant in five lumbar punctures made in each of two different cases. This, again, is too small a number of tests on which to base any conclusions, yet the constancy in all the determinations would make it worthy of further consideration. Should this difference hold good in all cases of meningitis, in which there is a definite inflammation of the meninges, it would be interesting to know whether or not it holds good in other abnormal conditions in which there is a lymphocytic cell count in the spinal fluid, for instance, poliomyeloencephalitis, in which the chief lesions are in the spinal cord. Assuming that it does not hold in infantile paralysis, it seems that it ought to be of value in the differential diagnosis between an early infantile paralysis and an early tuberculous meningitis.

Blood.—The lowest white blood count in the whole series was 8,500, while the highest was 48,000. The largest percentage (26.2) showed a leukocyte count between 10,000 and 15,000. There was no relation between the white blood count and the age or stage of the disease, although in the individual cases, in which more than one count was made during the progress of the case, the second count was usually lower than the first. Of the cases which showed a perfectly definite positive von Pirquet reaction, 66 per cent. had an average white count of 13,000, which is somewhat below the average count. Twenty differential counts are given in the accompanying table.

DIFFERENTIAL COUNT IN TWENTY CASES

No.	W. B. C. No.	Polymorphonuclears Per Cent.	Mononuclears Per Cent.	Transitionals Per Cent.	Eosinophils Per Cent.
1	26,800	74	25	1	0
2	16,400	41	59	0	0
3	14,000	65	33	1	1
4	9,800	55	44	1	0
5	33,600	90	10	0	0
6	10,500	72	28	0	0
7	9,500	79	21	0	0
8	25,000	57	38	4.5	0.5
9	22,400	90	10	0	0
10	13,200	80	20	0	0
11	25,700	85	15	0	0
12	15,600	80	20	0	0
13	11,000	68	32	0	0
14	21,000	84	16	0	0
15	26,300	80	19	0	1
16	15,400	54	45	0	1
17	30,800	75	25	0	0
18	36,000	74	26	0	0
19	14,000	69	31	0	0
20	41,700	90	10	0	0

The fact that in these twenty cases the eosinophils were practically absent, and also that these patients died, bears out the contention of

Swan and Karsner[6] that the disappearance of the eosinophils from the circulating blood may be regarded as an unfavorable prognostic sign. It is unfortunately impossible to give the differential counts of cases that have recovered from this almost invariably fatal disease.

Von Pirquet Reaction.—Of the cases in which a von Pirquet test was done, 63 per cent. gave a positive reaction, 25 per cent. a negative reaction, and 2 per cent. are recorded as suggestive. There was no relation between this reaction and the sex. All but three of the patients who were positive to tuberculin had had some previous illness, 35 per cent. having had measles, and 10 per cent. chicken-pox. All but five had comparatively high white blood counts. The relatively high number of positive tests obtained, and the exceedingly high number obtained by Holt and other observers, seem to be contrary to the established teaching that miliary tuberculosis, of which tuberculous meningitis is an example, is "often negative when the tuberculosis is of the miliary type."[7] Quite frequently, if the first test is negative, a second or third may prove positive. On one particular patient several tests were made on different parts of the body, and at different times, to see at what stage of the disease the body lost its reaction to tuberculin. The reactions on one side of the body were much more intense than on the other, and a test made seventy-two hours before death proved positive in twenty-four hours. Tests with bovine tuberculin were not done. There was no relation between a positive test and the age, for babies of 10 months responded just as frequently as children of 10 years. Nor was there any relation between a positive test and the length of time sick, for positive reactions were obtained all the way from three to fifteen days before the end. The state of unconsciousness did not seem to influence the test, for in over 35 per cent. of the positive cases the patients were unconscious on admittance.

History of Direct Exposure.—There was a definite history of a direct exposure to tuberculosis in 25 per cent. of the cases, no known exposure in 73 per cent., and not recorded in 2 per cent. Of the patients directly exposed, 46 per cent. had had no previous illness, 23 per cent. had had measles, 15 per cent. had had pneumonia and 15 per cent. had had chicken-pox. It seems that this percentage of exposure is indeed smaller than it should be, for if one is energetic enough, he can, in at least 50 per cent. of the cases, elicit some information which will throw light on some tuberculous focus in the family.

Conditions of Lungs on Entrance.—In 30 per cent. of the cases there were sufficient signs in the lungs to show that they were involved

6. Swan, J. M., and Karsner, H. T.: On the Behavior of the Eosinophilic Leukocytes in Cases of Pulmonary Tuberculosis, New York Med. Jour., 1907, lxxxv, 539.

7. Morse, J. L.: Case Histories in Pediatrics, 1911, p. 120.

in the tuberculous process. In 70 per cent. the lungs were negative. In two cases d'Espine's sign was recorded as present. In no case was there any record of any palpable mesenteric lymph-nodes. There was a slight general lymph nodular enlargement in many of the cases but it was not constant. A slight cough was present in 30 per cent. of the cases, absent in 57 per cent. and not recorded in 13 per cent. It was present in the last seven of the series, and probably is present in a greater percentage than reported, but the cough is often so slight that parents overlook it, and very frequently it is not asked about in the history taking.

Reflexes.—Eyes: In 71 per cent. of the cases the eye reflexes were abnormal, and in 29 per cent. the pupils were equal and reacted normally to light. In 26 per cent. the pupils were equal and reacted sluggishly; in 12 per cent. the pupils were equal but did not react to light; in 4 per cent. the pupils were irregular with no reaction to light; in 1 per cent. they were dilated and irregular, with no reaction to light; in 12 per cent. they were irregular but reacted sluggishly; in 7 per cent. they were dilated and reacted sluggishly; in 4 per cent. they were equally dilated with no reaction to light; in 5 per cent. the condition of the eyes was not mentioned. There was no definite time previous to the end when the eyes lost their reaction to light for as many as twelve to fifteen days before the end of the disease the eyes showed some irregularity, either in size or amount of reaction. Of those patients admitted twenty-four hours before the end nearly all showed an absence of pupillary reaction; all but three of those admitted forty-eight hours before the end showed marked irregularity, extreme sluggishness or no reaction; three were recorded as "equal and normal." Of those admitted three days before the end (15) the pupils were normal in only one; in 3 out of 9 patients admitted four days before the end the pupils were normal; 2 out of 11 patients showed normal pupils five days before the end; 2 out of 8 cases showed normal pupils six days before the end; 3 out of 8 cases showed normal pupils seven days before the end; 3 out of 6 showed normal pupils eight days before the end; 2 out of 7 had normal pupils nine days before the end; 1 out of 3 had normal pupils ten days before the end; 3 out of 6 showed normal pupils eleven days before the end; 2 out of 2 cases had normal pupils twelve days before the end; 5 out of 6 had normal pupils thirteen days or over before the end. In only a few cases (6) was the absence of the winking reflex noted, and that only in late cases.

Knee-Jerks: In over 30 per cent. of the cases the patellar reflexes were equal and active; in 21 per cent. they were absent; in 5 per cent. they were equal and sluggish; in 6 per cent. they were irregular; in 6 per cent. they were not mentioned; in 32 per cent. they were equal

and normal. There was no relation between the character or presence or absence of these reflexes and the sex or age of the patient. They were absent as long as eighteen days before the end, and were equal and active two days before the end.

Babinski's and Oppenheim's Signs: There was a positive Babinski sign in 21 per cent. of the cases, negative in 55 per cent. and not mentioned in 24 per cent. It is more frequent during the latter stages of the disease. The Oppenheim sign is elicited by rubbing the hand or an object along the internal aspect of the tibia and getting extension of the toes. It was obtained in 50 per cent. of the cases in which it was tried and negative in 50 per cent.

Ankle Clonus and Gordon's Sign: Ankle clonus was almost never present as an admitting symptom, though it was rarely reported as being positive. No mention was made as to whether it was a true or pseudoclonus. The Gordon sign, elicited by compressing all the muscles about the lower third of the tibia and getting extension of all the toes, was not mentioned in any of the admitting examinations, but in the last six cases of the series it was present late in the disease.

Abdominal and Epigastric Reflexes: These were positive in 14 per cent., negative in 17 per cent. and not mentioned in 69 per cent. The loss of these reflexes is also one of the late manifestations, usually occurring from three to four days before the end. In a few cases the abdominal reflex disappeared before the epigastric reflex.

Brudzinski's Neck Sign: This very important sign in meningitis, which Brudzinski[8] thinks is due to hypertonicity of the muscles of the legs and a physiologic predominance of the extensor muscles of the neck and back over the flexor muscles of the legs, was positive in 40 per cent. of the cases, negative in 35 per cent., and not mentioned in 25 per cent. Most of the cases which came in a week or ten days before the end showed the sign more frequently than those coming in early or late. The sign was more frequently present in children between the ages of 2 and 4 than before 2 years and beyond 4. It was practically absent after 5 years.

Brudzinski's Contralateral Sign: This sign is obtained by passively flexing one leg at the hip and getting an accompanying flexion of the other leg, this being known as the identical reflex; but if the leg is extended instead of being flexed, it is called the reciprocal contralateral reflex. Brudzinski[9] thinks this sign depends on the tendency to bilateral

8. Brudzinski, J.: Un Signe Nouveau sur les membres inférieurs dans les Méningites chez les enfants, Arch. de méd. d. enf., Par., 1909, xii, 745.

9. Brudzinski, J.: Experimentelle Untersuchungen über den Kontralateralen Reflex, und über das Nackenphänomen an den unteren Extremitäten, Wien. klin. Wchnschr., 1911, xxiv, 1795.

innervation of the muscles, which is more pronounced in infants, and that when the cerebrum is diseased there is a tendency to revert or bring out this bilateral innervation. The sign was reported as being positive in only eight cases, negative in eleven cases, and was evidently not looked for in eighty-six of the cases, as no mention of the sign was made. Inasmuch as this sign is a recognized one and very frequently present in meningitis, it seems that it ought to receive more recognition in the physical examination, and its presence or absence noted. In only two cases was there any note of a reciprocal contralateral being present.

Kernig's Sign: The Kernig sign was present in 27 per cent. of the cases, negative in 71 per cent., and not mentioned in 2 per cent. There was no relation between the patellar reflex and Kernig's sign, for it was present just as often in cases of exaggerated knee-jerks as it was in cases of sluggish knee-jerks.

Tache Cérébrale.—This symptom of cerebral involvement was mentioned several times, but in the majority of cases was not. Even though it does appear in other abnormal cranial conditions, yet its presence is a part of the symptom complex of tuberculous meningitis, and its presence or absence should be noted. The fact that, after stroking the skin, a red line appears is not sufficient evidence of a positive *tache cérébrale*. The line of redness should persist for some little time, and is often from one half to one inch in width.

Other Objective Signs.—Paralysis: 43 per cent of all the cases showed some kind of paralysis. Ocular paralysis (strabismus), single or double, was the most common, occurring in over 61 per cent.; ptosis of the eye-lids occurred in over 15 per cent.; nystagmus in 13 per cent.; facial paralysis in 4 per cent.; paralysis of the tongue in 2 per cent.; of the arms 2 per cent., and of the legs 1 per cent.

Rigidity and Retraction: In 70 per cent. of the cases there was rigidity of the neck and absent in 25 per cent. On an average, it was present five days before the end, and absent on an average of seven days before the end. In 19 per cent. of the cases the head was retracted; in 75 per cent. it was not. Retraction, like rigidity, is also a late manifestation, occurring, on an average, five days before the end. Only one case was reported in which opisthotonos was observed. A slight amount of head retraction will often be missed as long as the patient is observed when lying on the back. Tenderness on flexion of the head was mentioned in a few cases. In practically all of the late cases there was a definite spasticity of the limbs noted.

Bulging Fontanelle: The oldest patient presenting a bulging fontanelle was 18 months and the youngest was 5 months. Over 41 per

cent. of all the children under 18 months had a bulging fontanelle on entrance. One patient 8 months old and one of 12 months had depressed fontanelles three days before death, the duration of the disease in both cases being seventeen days. In one case (12 months) the fontanelle was level; in five cases the fontanelle was tense. Of all the patients presenting bulging fontanelles on entrance, all but three had had convulsions before entering the hospital. One patient had a bulging fontanelle sixteen days before the end, but had been sick three weeks before entrance. In no case was the posterior fontanelle reported bulging. In no case was it necessary to do a ventricular puncture in order to obtain fluid.

Convulsions.—Thirty-nine per cent. of the series had had convulsions before entrance, and 61 per cent. had not, the oldest child being 6 years while the youngest was 8 months. No child over 6 years had had convulsions. Not a few of the patients between the ages of 6 and 22 months had had no convulsions. One case of muscular twitching was reported. The presence of convulsions is also one of the later manifestations, appearing, on an average, four days before the end, and absent, on an average, seven days before the end. In every case but one which showed a bulging fontanelle there was a history of convulsions. Convulsions while in the hospital were rare.

Unconsciousness.—Forty-five per cent. of the patients were unconscious on admittance, and most of those (72 per cent.) were 3 years old or under. There seemed to be no relation between the stage of unconsciousness and the duration of the disease. Four patients admitted on the last day of the disease were unconscious; 10 patients two days before the end were unconscious; 4 patients three days before; 6 patients four days before; 5 patients five days before; 4 patients six days before; 2 patients seven days before; 1 patient eight days before; 4 patients nine days before; 2 patients ten days before. Of those unconscious, in over 80 per cent. there were obtained 20 c.c. or over of spinal fluid, while in 20 per cent. there was less than this amount obtained. The average white blood count of those unconscious was 18,000. Many of those unconscious had comparatively low white counts on entrance. The cell count of the spinal fluid in over 27 per cent. of those unconscious was below 100, while the others ranged between 100 and 300 cells per cm.

Subjective Symptoms.—In practically every history drowsiness, apathy, dulness mentally, languor, irritability and sleepiness were mentioned. In over 85 per cent. vomiting was recorded as an initial symptom, though it seemed to be more frequent at the beginning and the end of the disease rather than in the middle. In 10 per cent. there

was no vomiting. This vomiting, which is of central origin, had no relation to food and seemed to occur at any time of the day. In no case was there any record of projectile vomiting, such as occurs in the meningococcic form of meningitis.

Pain: Pain was a comparatively frequent symptom. The eyes were mentioned as being painful in 1 per cent., the teeth in 1 per cent., the ears in 2 per cent., the neck in 7.5 per cent., the head in 18 per cent., the iliac crests in 1 per cent. and the abdomen in 3 per cent. No pain was recorded in 47.5 per cent. Expressions of pain were evident in 7 per cent. It may be an early or late symptom. Headaches were present in 17 per cent. of the older children.

Other Minor Subjective Symptoms: Fifty per cent. of the cases gave a history of constipation; in 8.5 per cent. the movements were loose; in 23 per cent. they were normal; in 1 per cent. they were irregular. In a few cases picking the nose or eyelids or the bedclothes was noted. Grinding the teeth or gums, restless sleep, delirium, loss of sphincteric control, crying, capricious appetite and inability to swallow were frequently mentioned.

Urine.—In 18 per cent. of the cases acetone was present; a "very slight trace" of albumin was obtained in 7 per cent.; a "slightest possible trace" of albumin in 16.5 per cent. a "slight trace" of albumin in 2 per cent.; a "trace" of albumin in 2 per cent. In 30 per cent. the urine was negative, and in 26 per cent. the urine was not mentioned. Many of the "not mentioned" cases were those of patients who came in moribund and died before a specimen could be obtained. There was no relation between the urinary findings and the sex, white blood count, age or character of the spinal fluid.

Pulse, Temperature and Respiration at the End.—In 41 per cent. there was a final rise of all three curves; in 9 per cent. only a terminal rise of the pulse; in 12.5 per cent. there was a terminal rise of pulse and respiration; in 6.2 per cent. the temperature only rose; in 3.1 per cent. there was a final rise of the temperature and respiration; in 2 per cent. there was an elevation of the respiration, and in 12.5 per cent. there was no final rise of any of the three curves. There was no relation between the terminal rise and the von Pirquet reaction.

Terminal Bronchopneumonia.—From the clinical signs not verified by necropsy, 42 per cent. of the cases were diagnosed as terminal bronchopneumonia, while 61 per cent. of the necropsies done in cases in which death occurred from tuberculous meningitis showed bronchopneumonia to be present. Thirty-seven and five-tenths per cent. of the necropsies showing a terminal bronchopneumonia and 80 per cent. of the necropsies showing no bronchopneumonia were in females, which

would seem to show that a greater percentage of males develop a terminal bronchopneumonia than females. There seemed to be no relation between a terminal pneumonia and the previous illness, or cell count, or the number of lumbar punctures. There was just as large a percentage under 2 years of age who developed a pneumonia as there was under 2 years who did not. The white blood count at entrance was not increased in those cases which later developed a bronchopneumonia.

Relation of Pulse to Temperature.—The character of the pulse depended on the stage of the disease. Patients coming in comparatively early, for instance, two weeks before the end, showed a slow and irregular pulse, while in those in whom the disease was well advanced, the pulse was soft and rapid. Rarely a patient showed a slightly elevated and irregular pulse prior to the stage when it became slow and irregular. In most of the cases the pulse began to rise from one to four days before the temperature. On an average, the pulse began to rise five days before the end, while the temperature, on an average, began to rise two days before the end. A definite remission of symptoms, with a corresponding drop in pulse and temperature, occurred in 64 per cent. of the cases. This fact, as has often been noted, has deceived not a few who are not acquainted with this deceptive phase of the disease. Not infrequently, a patient who has been stuporous and comatose for days will brighten up and actually answer questions from twenty-four to thirty-six hours before death. It is interesting to note that at this stage of the disease there is also a drop in the globulin content and the cell count in the spinal fluid.

No record was kept as to the amount of sugar present in the spinal fluid. In several cases its presence was recorded, while in a few cases a decrease was noted. In no case was it recorded as being entirely absent. The eyes were examined in a few cases, but choroid tubercles were never seen, or if they were, they were never recorded. A moderate conjunctivitis was mentioned in a few cases, which usually came toward the end. No mention was made of any tubercle bacilli having been found in the conjunctival pus. The blood pressure was recorded in only four cases, in each of which it was lower than normal. The reaction of the spinal fluid was mentioned in only a few cases, it being alkaline in every case. The loss of the swallowing reflex occurred as much as ten or eleven days before the end.

Necropsy Findings.—The following list shows the frequency of the various pathologic lesions in eighteen necropsies performed in cases in which death occurred from tuberculous meningitis:

Bronchopneumonia 9
Miliary tuberculosis of the lungs ... 8
Miliary tuberculosis of the pleura... 2
Miliary tuberculosis of the spleen...10
Miliary tuberculosis of the liver..... 6
Miliary tuberculosis of the kidney... 6
Miliary tuberculosis of the spinal cord 3
Caseous tuberculosis of the liver.... 1
Caseous tuberculosis of the spleen... 1
Caseous tuberculosis of the lymph-nodes 7
Caseous tuberculosis of the lungs.... 6
Enlarged mediastinal lymph-nodes...11
Enlarged mesenteric lymph-nodes...10
Internal hydrocephalus (acute)..... 4
Internal hydrocephalus (chronic).... 1
Encephalomalacia 1
Miliary tuberculosis of the brain.....18
Solitary tubercle cerebrum 1
Solitary tubercle cerebellum 1
Solitary tubercle liver..... 1
Solitary tubercle spleen..... 1
Tuberculomata cerebellum (mult.).. 1
Tuberculomata cerebrum (mult.).... 2
Tuberculomata cord (mult.)..... 1
Chronic conglom. tubercle lungs..... 6
Dilated and hypertrophied heart..... 1
Subacute gastritis 2
Marked involution of thymus..... 1
Colloid struma (incipient)..... 1
Toxic nephropathy 2
Degenerative myocarditis 1
Chronic pachymeningitis external... 1
Miliary tuberculosis of the omentum 1
Miliary tuberculosis of the lymph-nodes 1
Tuberculous ulcerative enteritis..... 7
Tuberculous ulcerative colitis..... 2
Old pleuritis 3
Old apical lesions 3
Tuberculous ependymitis 2
Tuberculosis of the kidneys..... 1
Tuberculosis of the spine..... 2
Tuberculosis of the appendix..... 3
Tuberculosis of the thymus..... 2
Tuberculosis of the pleura..... 1
Tuberculous dermatitis 1
Tuberculous bronchi 1
Tuberculous ulcerative laryngitis.... 1
Tuberculous endocarditis 1
Tuberculous bronchopneumonia 3
Juvenile tuberculous focus of lung... 1
Localized encephalitis 1
Rachitis 2
Perisplenitis 1
Perihepatitis 1
Ulcerative stomatitis 1
Otitis media 2
Acute fibrous pleurisy..... 1
Polyp in duodenum..... 1
Slight cardiac dilatation..... 1
Hemorrhagic adrenals 1
Cystic ovaries 1
Bronchitis 1
Acute glomerular nephritis..... 1
Acute pyelitis 1

MICROSCOPICAL EXAMINATION

Kidney (cloudy swelling)..... 6
Heart (cloudy swelling)..... 4
Liver (fatty degeneration)..... 6
Liver (fatty infiltration) 4
Liver (cloudy swelling) 1
Spleen (acute hyperplasia)..... 2
Spleen (folliculous splenitis)..... 4
Lungs (emphysema) 1
Lungs (atelectasis) 1
Pancreas (cloudy swelling)..... 1
Colon (non-tuberculous ulcer)..... 1

Mortality.—In no case was there any recovery or remission from the disease. Various measures were tried, among which were large doses of hexamethylenamin and sodium benzoate, and inunctions of mercury. With excessive doses of hexamethylenamin there were no traces of formaldehyd in the spinal fluid.

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ACROCEPHALOSYNDACTYLISM *

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Acrocephalosyndactylism or acrosphenodactylism represents an unusual pathologic or teratologic type which was described at length by Apert[1] in 1906; recently Sterling[2] has summarized the reports of Apert and his predecessors in a brief review of the nine cases which constitute the basis for the literature of the subject to date. The condition conforms in general to the following description:

The skull is flattened posteriorly in such a manner that the external occipital protuberance is lacking; the squamous portion of the occipital bone is smooth and vertical; just above the lambdoid suture the skull bends forward and upward, reaching its maximum height at the bregma, where it meets, at rather an acute angle, the plane of the forehead and face. Although the frontal area has in general a vertical direction, there is a bulging in the transverse axis of the middle portion, which is separated from the arcus superciliares by a deep furrow. The frontonasal suture is widely open at birth and tends to remain so for the first three or four years of life or longer, while the lambdoid suture is usually solidly closed.

The face is modified more or less. The transzygomatic and transtemporal diameters are increased. The bridge of the nose is usually depressed and frequently there is exophthalmos. There is a horizontal shelflife projection from the right and left alveolar ridges into the mouth. These projections may even unite in their anterior portions to form a sort of gallery above which is the highly arched hard palate. The teeth are rather irregularly implanted.

In two necropsies on children, aged 2 and 3 months respectively, there was found marked shortening of the base of the skull in the sagittal diameter, involving especially the middle and posterior fossae. The sagittal suture was open from the lesser fontanelle to the root of the nose, being especially wide—about an inch and a quarter—in the frontonasal portion. Wormian bones were present in the posterior fontanelle. The frontal and parietal bones in both specimens presented numerous pits or elliptical depressions on their inner surfaces, where

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1. Apert, E.: Bull. et mém. Soc. d. hôp. de Paris, 1906, xxiii, 1310.

2. Sterling: Ztschr. f. d. ges. Neurol. u. Psychiat. (Referate u. Ergebnisse.), 1914, ix, 38.

the bone was extremely thin, and in a few places bony deposit over such areas was absent entirely. During life the bones of the vault of the skull could be indented with the finger. The base of the brain was similarly shortened in its anteroposterior diameter. The occipital lobes were quite small, so that the cerebellum was almost entirely uncovered and the whole organ appeared as though compressed, though no important convolutions were missing. There was no excess of cerebrospinal fluid.[3]

The hands and feet present a more or less complete syndactylism, which is always of the same type. In the hands the union is more intimate at the tips of the fingers than at the bases, and the hands,

Figs. 1 and 2.—Patient with acrocephalosyndactylism, front and side views of head and face.

therefore, are usually immobilized in the so-called *main d'accoucheur* position. Commonly the nails of the three middle fingers are fused. The thumb as well as the little finger may be free.

The toes are grown together into a homogeneous mass whose anterior edge is bordered by the nails of the fused toes. Frequently the great toe may remain free. In some cases the number of fingers or toes is reduced to four; in others the number may be increased to six or seven, even up to nine.

The etiology of acrocephalosyndactylism is unknown. There is nothing of a familial or hereditary character in connection with the condition. In some cases lues appears in the history; in others hydramnios

3. Wheaton: Tr. Path. Soc. London, 1894, xlv, 238.

was noted during pregnancy, but neither is of constant occurrence. The deformity of the skull is probably the result of the early closure of the sutures at the base and posteriorly. The syndactylism may be due to

Fig. 3.—Syndactylous hands of patient.

Fig. 4.—Feet of patient, showing fusion of toes.

amniotic adhesions, as suggested by Wheaton;[3] it has been suggested that there may be a trophic correlation between the faulty development of the base of the brain and that of the extremities. The condition does not necessarily shorten life, and the intelligence of the patient may not

be seriously impaired. The age of the oldest patient described was 21 years. The youngest patients were infants of a few months. No Roentgen ray studies have been reported.

REPORT OF CASE

The present case is that of a girl, 3½ years of age, who was received in the surgical service at the Presbyterian Hospital in November, 1914. She was brought to the hospital for advice concerning deformities of the head, hands and feet, which had been present since birth. Briefly, the history is as follows:

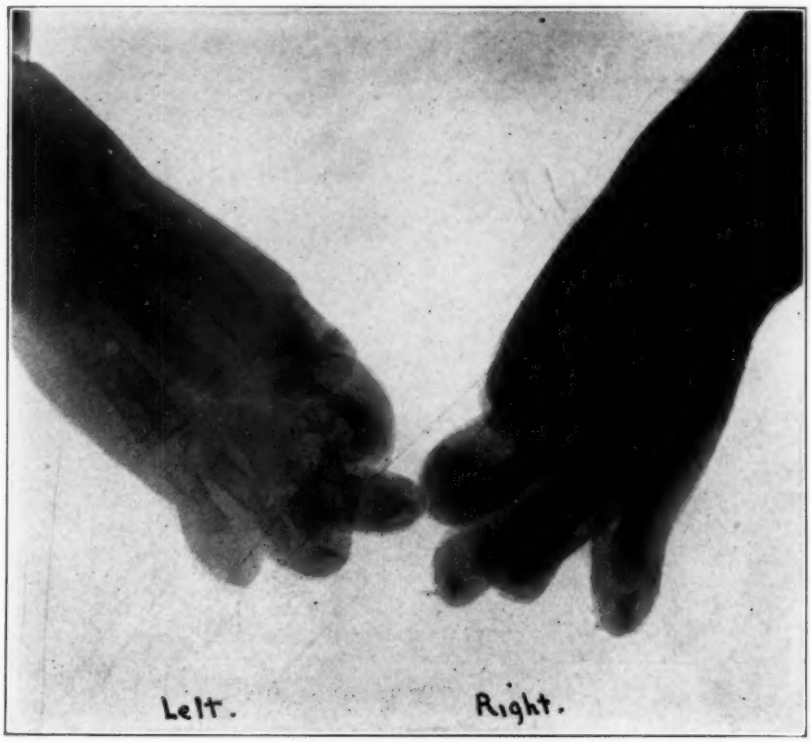

Fig. 5.—Roentgenogram of hands.

The child was born at term, normally, without instrumentation. She was a healthy, breast-fed child until the age of 3 weeks, when she was taken to the North Chicago Hospital for the first of a series of four operations on her hands. Under the influence of the operations and intervening attacks of bronchitis, after the first operation, chicken-pox after the second, measles after the third and whooping cough after the fourth, she remained a puny child until the middle of her second year, since which time she has been in good health and has developed rapidly. She walked at the age of 2 years and 2 months. The patient plays with the other children and the mother maintains that she is as active mentally as they. The father and mother are living and well. There are in the family three other children, aged 15, 13 and 5 years, respectively, who are said to be healthy. The mother had two miscarriages between the second and

fifth pregnancies. No Wassermann tests were made on members of the family other than the patient, in whom the test, using blood serum, was negative. The patient does not appear to be so bright mentally as the mother says she is. She seems to understand what is said to her but she never attempts to form sentences and very rarely attempts a word.

Physical and laboratory examinations disclosed no abnormalities excepting those in the head and extremities, to which exclusive attention may now be given. In general the head is relatively short in the sagittal diameters, and greatly lengthened in the frontomental, bitemporal and bizygomatic diameters. The shape is roughly that of a four-sided pyramid; the apex in the neighborhood of the lambda is rather bluntly rounded, dome-like; the base is represented by the face, which is somewhat diamond shaped, the angles being at the chin,

Fig. 6.—Roentgenogram of feet.

middle of zygomatic arches and the most prominent point on the smoothly rounded, rather bulging, open frontal suture. The face is flat and broad; the forehead high and precipitous, gently rounding laterally into temporal areas which slope outward and downward toward the prominent zygomatic arches. The superciliary ridges are highly arched and flattened. The roof of the orbit may be palpated for about 2 cm. posteriorly from its anterior border. There is marked proptosis; the eyeballs appear to be placed low in the orbits because of the excessive arch and flattening of the supra-orbital ridges. The bridge of the nose is low, but the end bulges out quite markedly. The lips and tongue are normal. There are ten fairly well formed but somewhat irregularly placed teeth in each jaw. The canines are not excessively prominent. The hard palate is of an extremely high arch, the sides coming together at an angle rather than an arch at the highest point, which is located very close to the junction with the soft palate. There is a horizontal shelf-like projection extending nearly the entire length of the alveolar margin of the superior maxilla. The general

shape and relations of this shelf may be ascertained by inspection of Figure 8, in which the condition is somewhat exaggerated. The hair of the head is abundant and of normal texture. The external ears are of normal shape and size.

Examination of roentgenograms of the skull disclosed the following points of interest:

The lambdoid and coronal sutures are closed. The sagittal suture between the nasion and anterior fontanelle is enormously dilated; posterior to the fontanelle it is closed. The sella turcica is markedly deepened and broadened. The orbits are considerably foreshortened and the roof of the orbit arches downward and backward quite sharply. Digital impressions are quite marked in the occipital and posterior portions of the parietal bones. These probably correspond to the pits or elliptical depressions described by Wheaton in his necropsy specimens.

The report of the opththalmologist (Dr. Darling) on the condition of the eye-grounds is as follows:

Media clear; nerve head gray and margin of disk blurred; could not measure, but looks as if there were some swelling of disk (edema); retinal vessels slightly tortuous.

The following measurements of the head were made:

Diameters—

Fronto-occipital	15.0 cm.
Bitemporal	12.5 cm.
Biparietal	14.5 cm.
Frontomental	17.5 cm.
Bizygomatic	13.5 cm.
Transorbital	10.0 cm.
Height of orbits	4.0 cm.
Length of frontal suture	9.0 cm.
Width of frontal suture	4.0 cm.

Circumferences—

Fronto-occipital	48.0 cm.
Occipitomental	51.0 cm.
Through gonion of each side and middle of cranium	25.0 cm.
Frontomental	48.0 cm.
Through lambda and supraorbital ridges	47.5 cm.
From lambda to glabella	28.0 cm.
Glabella to bregma	9.0 cm.
Auricular point directly across to auricular point of opposite side	34.0 cm.

The total length of the child was 84 cm.

The present condition of the hands is well shown in Figures 3 and 5. There is irregular fusion of the digits with some angular deformities, apparently due to cicatricial contractures. Practically all the joints in the fingers are stiff. Previous to the series of operations performed during the first year of life, all the digits of each hand were fused in the typical main d'accoucheur position. (Personal communication from Dr. Carl Beck who was the surgeon in charge at that time.) The fusion was so complete that but little knowledge could be gained as to the proper lines of cleavage between the phalanges. The separation was thus attempted, rather blindly, of necessity. The work was done with scissors. Roentgenograms made for the present study show conditions to be as follows:

Right Hand.—There are five metacarpal bones; the fourth and fifth are fused for their proximal two-thirds. The proper number of digits is present.

In the thumb there is only one shadow and no suggestion of division or joints. In the index finger there are three phalanges. The terminal phalanx is sharply flexed on the second, and appears to be ventrally curved like a hook. This hook may represent the missing phalanx from the thumb. The proximal interphalangeal joint is replaced by bone. The bones of the middle and ring fingers are closely apposed and seem to be continuous at the level of the proximal interphalangeal joints, while the joints are replaced by bone. The little finger is bent laterally at an obtuse angle, the apex of which is at the first interphalangeal joint; this joint has also been replaced by bone. The character of the joint changes suggests that they are the result of postnatal, inflammatory processes, rather than congenital anomalies.

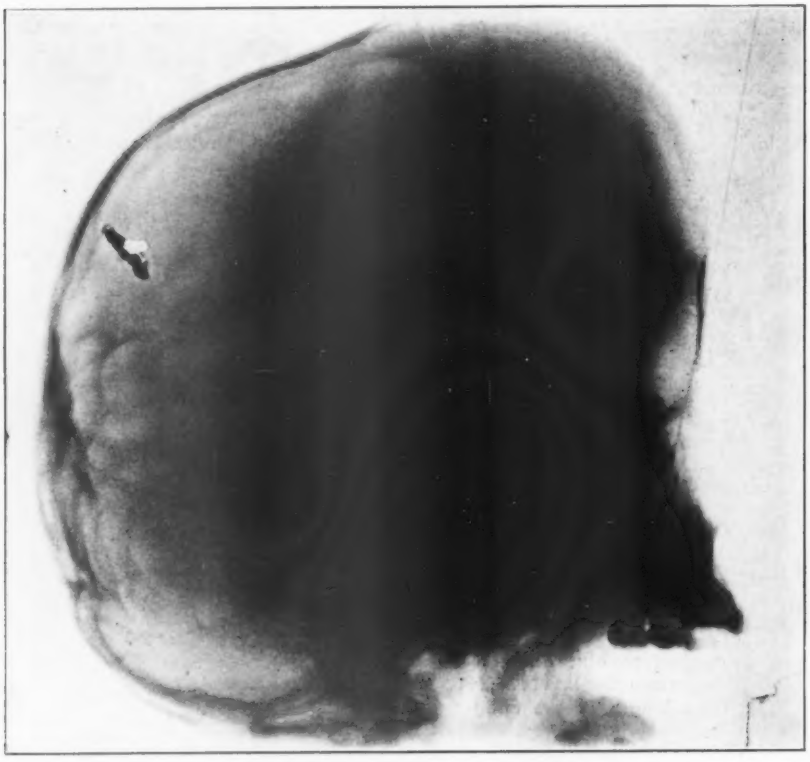

Fig. 7.—Roentgenogram of skull.

Left Hand.—There are five metacarpal bones. The fourth and fifth are fused proximally for one-half their length. There are no bone shadows in the thumb. The three middle fingers present bone shadows suggestive of a phalangeal complement for four fingers. This probably accounts for the bones missing from the thumb. These bones are irregularly fused into a bony plate with, apparently, obliteration of the proximal interphalangeal joints. In the little finger there are shadows of but two bones. There is a gap at the position which the middle phalanx should occupy.

The feet present no abnormalities until the metatarsophalangeal articulations are reached. At this point the webs and interphalangeal spaces fail to appear. Instead there is a uniform extension of the skin of the dorsum to within about

5 mm. of the anterior end of the foot, where it is crossed by a transverse row of nails, shorter and broader than normal, and contiguous with each other. There are five on the left foot and four on the right. On the plantar surface there is a shallow furrow running transversely across the foot just anteriorly to the metatarsophalangeal articulation; it extends dorsally over the medial margin of the foot in the position corresponding to the first phalanx of the great toe. The nail of the great toe of each foot faces laterally and slightly upward. The fleshy plantar pad faces medially and somewhat plantarward. The anterior margin of each foot slopes in a gradual convex curve backward from the medial to the lateral edge; it is slightly serrated, the number of serrations corresponding to the number of nails with one of which each serration is surmounted.

Fig. 8.—Sketch, indicating the deformity of palate.

The phalangeal portion is concave dorsally, convex plantarward; the dorsal concavity (and plantar convexity) is of low grade from the lateral edge medianward until the region of the junction of the first and second digits where there is a rather abrupt dorsal slant due to the marked rotation of the great toe on its long axis. This deformity prevents the patient from wearing shoes unless a large window is cut out over the great toe. There is no web between the digits; they are in close apposition and except at the ends, as mentioned, there is little indication of their presence. There is no independent motion of the portions corresponding to the individual digits, although the digital region as a whole possesses moderate mobility, especially on the little toe side.

Description of roengenograms of the feet:

In both feet the tarsal bones are normal.

In the right foot there appear to be six metatarsal bones; the first and second are fused distally. The first is incomplete proximally, while distally

it appears to extend without division or other trace of phalanges into the great toe. The fourth and fifth metatarsals are fused in their distal third. Phalangeal rudiments for six toes are present, including in the count the above mentioned distal portion of the first metatarsal.

The left foot is similar to the right except that the union between the first and second metatarsals is considerably more intimate, and the first metatarsal does not extend beyond the line of the metatarsophalangeal articulations. In addition the first and second phalangeal rudiments are closely united throughout their entire length and there is no fusion between metatarsals other than the first two.

Acrocephalosyndactylism seems to be a definite clinical entity presenting characteristic features which are the result of definite pathologic influences at work during intra-uterine life. The demonstration of the nature of these influences is not possible at present.

I am indebted to Dr. Peter Bassoe for the suggestion of the correct diagnosis in the case described above.

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CONGENITAL INTESTINAL OBSTRUCTION *

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Congenital mechanical obstruction of the bowels not due to a developmental defect is so rare a condition that the following case seems worth reporting.

Baby, male, 4 days old, first child, normal delivery, no bowel movement since birth. There had been no stain of meconium on the diapers, which had been observed carefully. Micturition was normal. On the second day the baby vomited a small amount of dark yellow material, rather suggestive of meconium. A second vomiting occurred on the third day, but not on the fourth, although the baby had nursed at four-hour intervals during the day. On the fourth day the abdomen was quite markedly distended and tense, so that no mass could be felt, although there seemed to be rather more resistance on the right side. There was slight dulness in the flanks, especially the right. Visible peristalsis was distinct and continuous. There was no intermittent crying suggestive of intussusception. A finger could be inserted 2 inches or more into the rectum and nothing could be felt except that the rectum seemed somewhat tighter than usual. Repeated soapsuds enemas brought no stain of meconium or blood—only very scanty mucus. Not over one ounce actually entered the bowel, the return flow pouring out around the catheter. At 2 p. m. on the fourth day the temperature was 102; at 8 p. m. it was 100.5. The child's color was good and its general condition good. The head was kept well retracted with moderate spasm. The chest was normal.

The child was taken to the hospital and the abdomen opened at 10 p. m. (fourth day). Immediately clear, slightly yellowish serous fluid poured out. On evisceration the condition was readily seen to be a complete twist of the mesentery of the small intestine with complete obstruction approximately at the middle of the small intestine. On untwisting, gas escaped through into the collapsed bowel beyond. The appendix, sharply coiled, and the ileocecal region were normal. The abdomen was closed and the child seemed to be in good condition. A catheter inserted into the rectum allowed a small amount of gas to escape but nothing else. During the night there were two good bowel movements of meconium. The next day the child seemed to be in fair condition in spite of a temperature of 104. There was a series of about ten bowel movements. Toward evening the color of the ears and the fingers became livid. The temperature, pulse and respiration continued high, the latter about 100. The head was retracted, the legs relaxed. The forehead was kept tonically wrinkled and the eyes wide open, suggestive of an abdominal facies, although the abdomen was soft and not unduly distended. The child died about thirty hours after the operation. Necropsy was not permitted.

When one is confronted with a case of intestinal obstruction at birth, the presumptive diagnosis is some developmental defect, the most common being intestinal atresia. The foregoing case is one of the rare instances in which a more remediable condition existed and is

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instructive from the point of view of prognosis and treatment of these cases as a class. A fairly careful review of the literature shows only four cases at all comparable to the one reported. These are referred to again in an excellent article by N. I. Spriggs,[1] perhaps the most comprehensive recent publication on the subject in English. A very complete bibliography is appended. The first of these cases was reported by Harrison Cripps in 1880, a case of congenital volvulus of the lower ileum. On rectal examination it was noted the little finger could be introduced a fair distance, but seemed to meet an obstruction suggesting a blind end. A congenital atresia was diagnosed and an inguinal enterostomy performed on the third day. There had been persistent vomiting and complete obstruction. The child died from peritonitis. At the necropsy the rectum, colon and about 2 feet of the ileum were found intact but extremely small, having about the caliber of a pipe-stem. A loop of ileum at this point was completely twisted on itself with distended bowel above. The undeveloped lower bowel was explained to be due to the absence of meconium from early fetal life and the consequent lack of stimulation to growth.

Newton Pitt in 1891 reported a similar case of congenital volvulus of the ileum, 2 feet from the ileocecal valve. It was also regarded as a case of imperforate rectum and inguinal colostomy performed. At necropsy a sharp line of demarcation could be seen between the strangulated and healthy bowel.

Spriggs also refers to Carwardine's case of congenital volvulus occurring at the point of Meckel's diverticulum with completely collapsed bowel below, and to another case by Eschback. None of these, however, had a complete twist of the mesentery as in the case reported above.

These cases, rightly considered, merely represent a more hopeful group of the general class of congenital intestinal obstruction. They may only differ in degree from the rest, since fetal volvulus, like other fetal accidents, such as intussusception, kinking, peritonitis, etc., have been pretty well shown to play a part in the causation of intestinal atresia. The etiology of the latter condition seems to be a very complex one. A large proportion of intestinal atresias occur at the location of some embryologic event such as the junction of the vitelline duct with the bowel or of the common bile-duct, which really indicates the point of the original evagination to form the liver, or the point of fusion between the hindgut with the proctodeum. There are many instances of atresia at other points, however. In fact, atresia of the alimentary tract may occur at any point or, although less frequent, at multiple points between the pharynx and anus. For this reason and

1. Spriggs, N. I.: Congenital Occlusion, Guy's Hosp. Rep., 1912, lxvi, 143.

from embryologic evidence, Kreuter and others hold to the theory that atresias are the result of the suspension of the process of canalization of the solid intestinal cord which exists in the early fetal life of vertebrates. Again, vascular accidents may be instrumental in this lack of development.

The symptoms of obstruction of the bowels at birth seem to be identical whether there is atresia or mechanical obstruction without atresia. Constipation usually is absolute from birth, because the condition usually dates from early fetal life before meconium has reached the lower bowel. There are a few cases on record, however, in which a few movements occurred just after birth and then stopped. In some a prenatal obstruction was obvious at necropsy. On attempting to give enemas it is frequently reported that the injection fluid returns directly around the catheter or at most 1 or 2 ounces are retained. This, together with the absence of any meconium or blood stains, has repeatedly led to the error of diagnosing a rectal atresia, which may be seemingly confirmed by an apparent obstruction of the rectum on digital examination. Perineal operations have been attempted under these conditions and valuable time lost without relieving the obstruction. If there is an imperforate rectum only, a bulging distended bowel should be palpated, especially when the child cries. If collapsed or small undeveloped bowel is encountered, there is probably atresia much farther up in the abdomen. In the first place, it should be remembered that rectal atresia practically always occurs within two inches of the anus.

As in acquired ileus, vomiting is a practically constant symptom and occurs early, usually the second day. The vomitus may contain meconium, bile or blood-stained mucus, the character of it giving some clue to the level of the obstruction. The child may nurse fairly well.

Distention nearly always occurs, but is not usually present at birth, although in a case reported by MacCallum it was so extreme as to interfere with parturition. This was due, however, to fetal peritonitis with many adhesions.

Icterus neonatorum has been mentioned as a symptom, but it would be difficult to say whether it is more frequent in these cases than in normal new-born infants. Visible peristalsis is of considerable aid especially when the obstruction is high and associated with gastric dilatation, which might be determined by percussion, skiagraphy or lavage. Free peritoneal fluid should also be examined for. Anuria has been described, even in cases in which there were no developmental defects in the urinary system. Convulsions have also occurred.

In the diagnosis it might bear repeating that too much attention should not be paid to a questionable stenosis of the rectum 2 inches or

more from the anus, because the lower bowel may be extremely small and undeveloped from a high obstruction. The possibility of general peritonitis must also be considered.

The prognosis in these cases is somewhat different from that of intestinal obstruction in adults. Serious symptoms develop comparatively less rapidly. The majority of patients will die in the first week, although many are on record living from fifteen to eighteen days. In general, the lower the obstruction the longer they will live. Cripps reported a case of imperforate rectum in which operation was refused on the third day. The child was brought back at the end of a month with little change in its condition in spite of fecal vomiting every two days. Spriggs quotes a case of a girl of 14 years with complete rectal occlusion, who vomited her feces every few days. A well-marked stenosis may cause little or no symptoms, but is probably the cause of some cases of Hirschsprung's disease later in life. It should be remembered that inspissated meconium may cause complete obstruction which may be fatal, or may be spontaneously relieved. There is a case on record[2] in which a child with congenital obstruction was in such desperate condition on the third day that operation seemed inadvisable. When it was 5½ days old, during a spell of crying, a mass of meconium was expelled, followed by a series of copious movements. The child fully recovered. For this reason it would seem justifiable to give gentle massage and enemas a fair test—for a short period of time, however, since the loss in weight is so rapid and the operative risk at best great. Spriggs states that in at least eighty-two cases of operation there has not been a single recovery, although two patients lived a couple of weeks. Most of the cases, however, had extensive anastomoses or enterostomies. The practical point is that conditions such as volvulus, intussusception, etc., at or before birth cannot be distinguished from intestinal atresia, which is hopeless without operation and operation for the former conditions might succeed in a fair proportion of cases.

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2. Med. Rec., 1899, lvi, 133

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